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STUDIES ON Rh ISO-IMMUNIZATION.

By N. R. HENRY and R. T. SIMMONS,

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THE reports on iso-immunization by the Rh factor both through pregnancy and through repeated blood transfusions have formed an ever-increasing part of medical literature in recent years. In 1936 several workers reported proved cases of transfusion reactions following transfusions of blood from individuals of the same A-B-O blood group. In 1939 Levine and Stetson[1] reported a reaction from a transfusion of homologous blood during the puerperium and suggested that the mother was immunized by the fœtus with the formation of atypical antibodies.

Soon afterwards, Landsteiner and Wiener[2] discovered and named the Rh factor, the discovery being the result of planned and careful research; the interest of all blood workers became centred on this factor, and then, soon afterwards, on the new phenomenon of iso-immunization within the human species.

Wiener and Peters,[3] in a study of cases of reactions following repeated transfusions of homologous blood, showed that the patients were Rh negative and that they formed atypical antibodies which had the same specificity as the Rh antibody. Iso-immunization in pregnancy due to the Rh antigen was successfully demonstrated by Levine, Katzin and Burnham,[4] and was soon confirmed by many other workers. The published data thus proved that the Rh factor was iso-antigenic in a dual role, (i) by immunization by repeated blood transfusions, and (ii) by immunization by the fœtus in pregnancy.

The Rh Antisera and Rh Nomenclature.

It soon became evident, however, that all human Rh antiserum was not identical in its specificities, but showed individual variation.[5] Knowledge and experience with Rh antiserum rapidly increased, and at this time another variety of Rh antiserum was found which gave approximately 70% of positive reactions (instead of the usual 85%) with the blood of white subjects.[6] Later a third variety of Rh antiserum was found by Wiener and Sonn[7] which gave approximately 30% of positive reactions with blood samples from American white individuals, and thus the whole conception of the possible Rh types in blood underwent a period of evolution. Various nomenclatures for the Rh system have been suggested, and the question of nomenclature is at the present time being considered by an international committee. The nomenclature used in this paper is that suggested by Wiener.[8]

The three types of Rh antiserum he designates as anti-Rh_0 (85%), which is the standard serum, anti-Rh′ (70%) and anti-Rh″ (30%). By the use of these three types of serum the Rh type can be divided into no less than eight different subtypes. These types are determined by six allelic genes—namely, Rh_0, Rh′, Rh″, Rh_1, Rh_2 and rh. Combinations of these genes give rise to eight different phenotypes, all of which have actually been demonstrated. These are Rh_0, Rh_1, Rh_2, Rh_1Rh_2, Rh′, Rh″, Rh′Rh″ and Rh-negative. Type Rh_1 contains both Rh_0 and Rh′ antigens, and so may be written Rh_0' or Rh″. Similarly, type Rh_2 may be written Rh_0'' or Rh‴.

Fisher (quoted by Race[10]), in England early in 1944, proposed another scheme of nomenclature for the Rh types and showed the relationships between the various types of Rh antiserum and the Rh genes. The brilliance of this theoretical scheme becomes obvious when it is realized that it postulated the presence and serological reactions of the two rare genes Rh_6 and Rh_7, and that it postulated the

presence of six types of Rh antiserum, of which four had been discovered at the time. The Rh_1 gene has since been discovered[37] and found to conform in every detail to the expected scheme, whilst one other of the types of antiserum has since been discovered by Mourant,[38] and found to conform completely to Fisher's postulated scheme. Fisher denoted the three forms of allelomorphic antigens which make up each gene as C, c, D, d, and E, e, and the antibodies specific for these antigens were denoted by the analogous Greek letters. The following table (Table I), modified from Fisher, shows the completness of the scheme.

Wiener,[14] in a recent paper dealing with the Hr blood factors, has suggested that the antibodies be known as anti-Hr′, anti-Hr″ and anti-Hr_0 in a manner similar to their corresponding Rh antiserum.

Recognition of the Rh Genotypes.

When it was thought that the sixth type of serum of the series had actually been discovered, Fisher[20] published the following concise statement concerning the recognition of the various Rh genotypes.

> With the complete set of six antibodies now apparently discovered, the recognition of rare genotypes will be immensely easier. All the homozygotes of the seven known allelomorphs can be distinguished unambiguously, and nine of the heterozygotes, namely (omitting h's), R_0r, R_0R_2, R_0R_1, R_2R'', R_2R_1, $R''r$, $R'r$, R_1R_2, R_1R'. Three pairs of heterozygotes are indistinguishable:
>
> R_0R'' R_0R' R_2R_2
> R_2r R_1r R_1R_2
>
> These leave little practical doubt, since in each case the genotype printed below is about 1,000 times more frequent than that above.
>
> Three more heterozygotes are capable of confusion only with a heterozygote involving R_y, which has not been discovered, and which is unquestionably very rare:
>
> R_yR_2 R_yR_1 R_yr
> R_1R'' R_2R' $R'R''$
>
> It is amongst these that R_y may be looked for.
>
> Finally a group of three known genotypes, and one involving R_y, should react positively with all six reagents. These with their approximate frequencies in the British population are shown below:
>
> R_1R'' R_2R' R_2r R_0R_y
> 0·8% 0·3% 0·4% very rare indeed
>
> Thus it is only amongst these last that genotype recognition will have to rely on pedigree evidence. The interpretation of pedigree evidence also will now be greatly facilitated.

The Rh "Blocking" or "Incomplete" Antibody.

Wiener[9] has described a different type of Rh antibody which he called the "blocking" antibody. Race[10] described the same antibody and called it an "incomplete" antibody. This antibody is capable of combining with the specific antigen (red cells) without any visible sign of the reaction having occurred. It was found to combine with the antigen (test cells in saline solution), but was incapable of completing the second stage of the agglutination reaction *in vitro*—that is, agglutination. However, that a combination has occurred may be shown by adding to the cell-serum mixture a known potent anti-Rh_0 agglutinating serum suitably diluted.[1] Since the antigens of the cells have already entered into combination with (or been coated by) the blocking antibody, none will be available for the agglutinating antibody. Therefore no agglutination will take place, whereas normally the cells would be strongly agglutinated. To date these "blocking" or "incomplete" antibodies found by most workers have been of specificity anti-Rh_0, but Diamond and Denton[35] have recently reported two cases of blocking antibody of both anti-Rh_0 and anti-Rh′ specificity, and in another report[39] stated they had found blocking antibodies of specificity anti-Rh′, anti-Rh″ and anti-Hr, but these are rare.[2] By the use of the ordinary agglutinating reaction together with this blocking test, Rh antibodies can be demonstrated in about 92% of the cases of sensitization.[34]

The Conglutination Test for Rh "Blocking" Antibody.

A further test to detect Rh sensitization has recently been described by Wiener[33] and is known as the conglutination test; but there is some doubt whether this is a true conglutination reaction. This test is exceedingly simple and consists of testing the red cells suspended in compatible human serum (known to be free from non-specific effects) instead of in citrate or saline solution, and any dilutions of the test serum (for titre determinations) are also made with this normal homologous serum instead of saline solution. The conglutination test detects "blocking" as well as agglutinating antibodies and gives visible agglutination with both types. The test may be performed at room temperature or 37° C., and the strength of cell suspension used is 2%. Wiener has postulated that the visible agglutination given by "blocking" antibody is due to some colloid in the serum or plasma used for the cell suspension.

[1] The words "suitably diluted" cannot be too strongly emphasized. The serum should be used at such a dilution that it causes "+++", not "++++", agglutination of the test cells, because the use of potent anti-Rh_0 serum used undiluted does give agglutination in the presence of "blocking" antibody.

[2] Callender, Race and Paykoc, *British Medical Journal*, July 21, 1945, at page 83, also reported having found the "incomplete" form of the St antibody.

TABLE I.

Reactions of the Six Postulated Types of Antiserum with the Eight Rh Genes. (Modified after Fisher and Others.)[1]

Name of Serum.		Anti-C. Anti-Rh′.	Anti-c. Anti-Hr′. (Standard.) (= St.)	Anti-D. Anti-Rh_0. (Standard.)	Anti-d. Anti-Hr_0.	Anti-E. Anti-Rh″.	Anti-e. Anti-Hr″.
Antibody present		Γ	γ	Δ	δ	H	η
Percentage reactions with the blood of white individuals		70%	80%	85%	65%	30%	96%
The genes and their reactions.	Rh_2 CDE ..	+	−	+	(−)	+	−
	Rh_1 CDe ..	+	−	+	(−)	−	+
	Rh_y CdE ..	(+)	(−)	(−)	(+)	(+)	(−)
	Rh′ Cde ..	+	−	−	(+)	−	+
	Rh_2 cDE ..	−	+	+	(−)	+	−
	Rh_0 cDe ..	−	+	+	(−)	−	+
	Rh″ cdE ..	−	+	−	(+)	+	−
	rh cde ..	−	+	−	(+)	−	+

[1] Neither the anti-d serum nor the Rh_y gene has yet been found. The predicted reactions are shown in parentheses.

The Open Slide Test for Rh Agglutinins.

The conglutination test is a modification of Diamond and Abelson's[28] rapid slide test for the detection of Rh agglutinins, or compatibility test. These authors first described this rapid and accurate slide test, which is so simple that it can be performed in the smallest laboratory, and which also detects "blocking" as well as agglutinating antibodies. The test consists of adding 0·2 millilitre of fresh oxalated blood of known Rh-positive and Rh-negative types to 0·1 millilitre amounts of the test serum on ordinary open slides. The mixture is rocked and slightly warmed. Visible agglutination usually appears within one minute and always within three minutes, while the Rh-negative cells act as controls. A thick suspension of blood in plasma is used containing 40% to 50% of cells. The authors postulated that the excessive number of red cells used in the test probably absorbed the "blocking" antibody and left the agglutinating antibody free to act. Wiener,[13] however, found that the reaction could be brought about by serum which contained "blocking" antibody without any agglutinating antibody, and also with much weaker cell suspensions, provided they were suspended in serum or plasma. The success of the test appeared, then, to depend on the avoidance of any crystalline solution in the mixture rather than on the concentration of cells. The disadvantages listed against Diamond and Abelson's open slide test are that the large area exposed on the slide is conducive to drying, that the high concentration of cells favours the formation of rouleaux (the addition of saline solution after agglutination has taken place breaks up the rouleaux), and that the thickness of the mixture makes microscopic examination of the cells difficult.

For finer work and titre determinations Wiener's conglutination test is preferable. By the use of these two tests antibodies have been demonstrated in practically 100% of cases in which Rh sensitization has occurred. These tests, however, are not successful with cells suspended in citrate or saline solution.

The Albumin Test for Rh "Blocking" Antibody.

Diamond and Denton,[25] in a later report, state that albumin, either human or bovine, is the most satisfactory suspension medium for the test cells, and that excellent results are obtained provided the final concentration of the albumin is kept above 13%. To this end they use 20% albumin (prepared from human or bovine crystalline albumin, presumably in saline solution) as the diluent. Albumin possesses the great advantage that it is free from non-specific effects. As in Wiener's conglutination test, the strength of the cell suspension used is 2%, and the test is carried out by the tube technique.

Diamond and Denton have suggested that the simple agglutinating antibodies appear early in the process of sensitization, and that the "blocking" type appears only as the result of more severe or prolonged sensitization. By titrations of a test serum they claimed to assess with some degree of accuracy the degree and period of sensitization.

One of us (R.T.S.), prior to the publication of the work on albumin by Diamond and Denton, had tested a number of fluids to determine their suitability as suspending media for cell suspensions; these fluids included isotonic starch, gelatin, pectin, globulin, fibrinogen and albumin. The most satisfactory medium found was 25% human albumin in buffered diluent, a product prepared for human injection by the Massachusetts Department of Public Health, Boston. A sample of 25% human albumin in saline solution prepared at the Commonwealth Serum Laboratories by Mr. Gordon Ada gave similar results. The tests were performed on slides with 5% cell suspensions, and also in tubes with 2% cells.

Antigenicity of the Rh Genes.

Wiener[11] has pointed out that the gene Rh_0 is the most antigenic by far of the Rh factors, Rh″ being the least antigenic and Rh′ occupying an intermediate position. The following quotation is from Wiener.[11]

> Therefore, it would be expected that if, for example, an Rh-negative woman bears a type Rh_1 fœtus and becomes sensitized to the Rh factor, her serum should either contain anti-Rh_0 alone, or anti-Rh_0 and anti-Rh′ together, but not anti-Rh′ alone. Actual observations fully satisfy these expectations; the cases where the maternal sera appeared to contain anti-Rh′ alone all proved actually to be examples of anti-Rh_0′ sera with anti-Rh_0 "blocking" antibodies.

The purpose of this paper is to report the investigations carried out in the case described here, which bear out the foregoing observations. In addition, this case has certain features which afford us the opportunity of stressing, in particular, two warnings to those engaged in the practice of blood transfusion: (i) Patients likely to receive repeated blood transfusions should be Rh tested and, if found to be Rh-negative, should receive only Rh-negative blood. (ii) The use of donors who have been in a malarious area should be avoided whenever possible. This is of great importance in view of the fact that many of our potential donors for years to come will consist of subjects who have been in such areas.

Report of a Case.

Mrs. M.H., aged forty-nine years, was first admitted to the Brisbane General Hospital in 1932, complaining of severe epistaxes and with a history of secondary anæmia. Previously in North Queensland she had been treated for pernicious anæmia by liver extract injections without effect. She had had at the Brisbane General Hospital cauterization of her nasal septum without relief. An erythrocyte count on September 8, 1932, showed her blood to contain 3,150,000 erythrocytes per cubic millimetre; her hæmoglobin value was 35% and her colour index 0·6. On September 10, 1932, a transfusion of fifteen ounces of blood from an unknown (hypertensive) donor was attempted, but only four and a half ounces were introduced. On September 15, 1932, she was successfully given a transfusion of eighteen ounces of blood from a donor (L.H.), who has since left this country. The patient's nasal septum was recauterized, and she was discharged from hospital on October 10, 1932, with an erythrocyte count of 4,270,000 per cubic millimetre and a hæmoglobin value of 75%.

She was admitted to the Ipswich Hospital in 1939, and was given a transfusion of 300 millilitres of blood from her son (C.H.), apparently without any untoward effect.

She was again admitted to the Brisbane General Hospital on June 22, 1945, complaining of palpitation, breathlessness, and a tightness and pain in the chest on the slightest exertion. She had been investigated privately and a diagnosis of hereditary hæmorrhagic telangiectasia had been made. On her fingers multiple telangiectases were present, and examination of the tongue revealed more telangiectases. The patient had suffered from chronic epistaxes. The erythrocytes numbered 2,940,000 per cubic millimetre, and her hæmoglobin value was 40%. Occult blood was present in the fæces.

In view of her anæmia, on June 23 a blood transfusion was given into the left saphenous vein. Her veins were extremely thready and deep, and venepuncture was difficult. For this transfusion her son (C.H.) and his friend (A.W.) were chosen after the cross-matching tests by the ordinary open slide method had proved them satisfactory. Both donors were servicemen. Five hundred millilitres of blood were removed from each donor, and the transfusion was commenced at 2.15 p.m. from donor C.H. At 7 p.m. during the course of transfusion of the second 500 millilitres of blood, the rate was slowed because the patient's pulse rate was steadily rising. At 8 p.m. the transfusion was stopped, as the pulse rate was still rising and the patient was "feeling dreadful". She had received 600 millilitres of blood in all. Her urinary output was maintained, however, and on June 25 she was feeling better. Her hæmoglobin value on this date was only 44%. On this date also her leucocyte count was 20,000 per cubic millimetre.

By June 28 she was complaining of headache and pains in the back and shoulders. She felt cold and shivering, and had photophobia. When the patient was questioned it was found that the second donor used on June 23 (A.W.) had had a malarial attack a few hours after he had donated the blood. Moreover, he had had other attacks previously. A blood film from the patient showed the presence of numerous *Plasmodium vivax* (benign tertian) parasites. Antimalarial therapy was instituted. The patient at this time was passing bloody stools. Because of this and because of her low hæmoglobin value (30%), on July 3 a further blood transfusion was arranged. The reaction to the previous transfusion not having been reported to the pathology department at the hospital, the ordinary open

slide method was again used for this cross-matching test. Blood from the blood bank (donor O.O.) was used for this transfusion after cross-matching tests had given satisfactory results. The transfusion of 500 millilitres of blood was commenced at 5·30 p.m. The patient's condition deteriorated, and she almost continuously passed semi-solid black fæces. Her temperature rose to 102° F., and the transfusion was stopped after 400 millilitres had been given.

The hæmorrhage into the bowel still continued, and the patient showed no improvement. This second reaction was reported and a request was made for the patient's blood to be Rh tested. She proved to be Rh-negative and her serum now contained weak anti-Rh agglutinins. Two group O Rh-negative donors were immediately obtained, and their blood was matched with the patient's by Levine's technique (thirty minutes' incubation at 37° C.). The blood samples proved to be completely compatible; 600 millilitres of Rh-negative blood were administered on July 4 without reaction, and the patient felt much improved. Vomiting and bowel movements had ceased. Her hæmoglobin value on July 5 was 35%.

Unfortunately, samples of the patient's serum taken before the first and second transfusions (June 23 and July 3, 1945) had been discarded, so that no tests for Rh antibodies were possible on these specimens. On July 8 the patient had a severe epistaxis and some vomiting. Her hæmoglobin value on July 9 was only 25%. The blood of two more group O Rh-negative donors was cross-matched with the patient's by Levine's technique, and 1,000 millilitres of blood were given into the left forearm, again without reaction and with obvious benefit to the patient. Her hæmoglobin value on July 12 was 45%.

On July 10 a further specimen of blood was taken for tests for Rh antibodies. The serum proved to contain anti-Rh agglutinins of titre 512 when tested against Rh_1 cells. The first two donors (C.H. and A.W.) were later recalled for Rh typing, and the results are shown in Table II.

Apart from a troublesome cough, the patient felt much better, and it was decided to give her another blood transfusion further to improve her condition. Rh-negative blood was again used on July 24, and the hæmoglobin value rose to 48% on July 27. (It had been 40% before the transfusion.) In spite of this increase, the patient complained of headache and vomited continuously. She was nauseated for four or five days after the transfusion. No hæmolytic phenomena developed, and this phenomenon might be explained by the fact that the patient suffered chronically from headaches and vomiting, and that she was also "psychologically sensitized" to, and detested, blood transfusions.

Her hæmoglobin value remained around 45% to 50% until the end of August, when it had again fallen to 30%. By September 6 it was 25%, and a further transfusion was arranged. As every available vein had been used, the sternal route was chosen. Five hundred millilitres of Rh-negative blood were administered by this route on September 7, 1945. The transfusion was reactionless, except for pain at the needle site. Her hæmoglobin value rose satisfactorily as a result.

A severe epistaxis lasting one hour, however, necessitated another transfusion, which was given by the sternal route on October 10. Response was satisfactory. Her condition remained more or less *in statu quo* for some time, and by oral administration of "Ferro-Hepamult" she succeeded in reaching a hæmoglobin value of 65% on December 7. She was discharged from hospital on December 22, as she desired to be home for Christmas, and, moreover, she was feeling quite well. She was, however, again admitted to hospital on January 18, 1946. Six days previously she had passed a quantity of blood *per rectum*, and two days later she had vomited a pint of heavily blood-stained fluid. Her hæmoglobin value on her admission to hospital was 30%. On January 2 she was given a transfusion of 1,000 millilitres of Rh-negative blood. She was still in hospital at the time this paper was prepared.

Material and Methods.

The first sample of blood for an Rh investigation from this patient, of blood group A, was received on July 4, 1945—one day after the second transfusion reaction had occurred. Washed cells suspended in saline solution were tested against an Rh-testing serum by the standard tube technique and were considered to be Rh-negative. The patient's serum was then tested for the presence of atypical antibodies by the tube technique against known Rh-positive cells of group O, and the serum was found to contain weak Rh antibodies. Titre determinations on a sample of serum obtained one week after this transfusion reaction showed the serum to have an agglutinating titre of 512 when tested against group O Rh_1 red cells. The patient's cells were also confirmed as Rh negative. No further investigations were made on this sample of serum until some eight weeks later, when it was tested for Rh specificity by one of us (R.T.S.), using cells of known Rh subtypes and a slide technique. It was found that the serum was a specific and potent anti-Rh′ (70%) serum and contained anti-Rh_0 "blocking" antibody.

It was then decided: (i) to test the available donors for their respective Rh types; (ii) to obtain progressive samples of the patient's blood and follow the changes in Rh-agglutinating and "blocking" titres; (iii) if the Rh-agglutinating titre had fallen in the meantime, to attempt, if possible, reimmunization with small intravenous doses of cells of group O Rh′.

Samples of blood (two drops) were collected into amounts of one millilitre of a modified Rous and Turner solution from the patient and donors, and were sent to Melbourne by air-freight. (We are grateful to Dr. Yum, who kindly collected blood from one donor at Gladstone, Queensland, and also to Dr. G. A. Kelsall, Perth, Western Australia, who sent us a large supply of blood of type Rh′ for experimental purposes.) The Rh types were determined on slides as described by Simmons *et alii*.[(10)] The cells were tested directly from the Rous and Turner solution, as this procedure is considered preferable to the use of cells washed in saline solution, and is much less bother and less time-consuming.

The anti-Rh titre determinations of the patient's serum were performed by serial dilutions in small (two inches by 0·25 inch) test tubes (parallel tests were also performed on slides), and throughout the experiments the same control test cells of group O MN Rh_1 were used, except where otherwise indicated. Three different diluents—saline solution, group AB serum and 25% strength human albumin—were used in diluting the patient's serum, while the test cells were suspended in (*a*) saline solution, (*b*) Rous and Turner solution, (*c*) group AB serum, (*d*) 25% strength albumin and, finally, (*e*) cells washed three times in saline solution which were used neat and added with a platinum loop to give the usual concentration.

Results.

The results of the Rh type tests on blood samples from the available donors are set out in Table II. The patient's blood was also confirmed as Rh negative. The tests were made with anti-Rh_0, anti-Rh′, anti-Rh″ and anti-Hr serum.[1]

The four transfusions received by the patient from 1939 to 1945 were all from subjects of blood type Rh_1 (Rh_0′). The patient, as has already been stated, produced anti-Rh serum of type anti-Rh_0′; but the anti-Rh_0 "blocking" antibody converted the serum into one with a specificity of anti-Rh′ (70%), which is in agreement with the statement made by Wiener.[(11)] As Rh antiserum with a specificity of anti-Rh′ is rare, another "trial bleed" was obtained from the patient, with a view to obtaining blood if the titre was still satisfactory. It was our intention to replace what-

TABLE II.
The Rh Types of Mrs. M.H.'s Donors.

Donor.	Date of Blood Donation.	Rh Type.[1]
L.H.	September 15, 1932.	Not available.
C.H.	January 13, 1939. June 23, 1945.	Rh_1.
A.W.	June 23, 1945.	Rh_1.
O.O.	July 3, 1945.	Rh_1.

[1] The three blood samples tested gave positive results with anti-Hr′ serum (Wiener and Vogel specimen).
The possible genotypes are: R_1r (most common), R_1R_2 and R_2R'.
As the patient was of genotype rr, the son C.H. would therefore be of genotype R_1r.

[1] The anti-Rh″ and anti-Hr′ specimens of serum used were samples kindly supplied by Dr. A. S. Wiener, New York.

ever blood was taken with double the amount of blood from an Rh-negative donor. This "trial bleed" was obtained on September 9, 1945, sixty-eight days after the transfusion reaction; but unfortunately the agglutinating titre had dropped to two, so that the serum was useless as anti-Rh' typing serum. The "blocking" titre, however, was still approximately 512 when the serum was tested by Wiener's blocking test. The test cells used were of group OM Rh_2. The sample of serum was then submitted to various tests, as shown in Table III.

The titre readings were, in general, made after two hours' incubation at 37° C. Some comparative tests were made at room temperature, but incubation at 37° C. was found more satisfactory.

This serum sample, obtained from the patient sixty-eight days after the last transfusion reaction, shows (Table III) only a trace of Rh-agglutinating antibody, which might easily be missed in a routine test. There are a number of interesting features in this table.

1. The Rh "blocking" antibody titres obtained by Wiener's conglutination test and by Diamond and Abelson's albumin test were approximately the same—8,000 to 16,000; but when combinations of the two tests were employed, the titre was shown to be 262,000. With Wiener's original blocking test the titre was only approximately 512.

2. When the test cells were suspended in albumin and a saline diluent was used for the patient's serum, it was surprising to find that the blocking titre was 16,000. With cells suspended in serum tested against the same saline series the titre was 128, but with cells in saline solution the titre was only two. (This figure is the actual Rh-agglutinating titre.)

3. Cells thoroughly washed with three lots of saline solution and used neat in tests against the patient's serum diluted with serum, and against the patient's serum diluted with albumin, gave titres of 16,000 and 2,000 respectively. The lower titre of 2,000 was probably due to the difficulty of obtaining complete agglutination in 25% strength albumin, which is extremely viscid. The neat cells were added to the tests with a 30 gauge platinum loop to give the usual cell concentration. This test follows work carried out by Simmons and Christie (unpublished observations).

4. It was unexpected also to find that cells in saline or Rous and Turner solution tested against the patient's serum diluted with albumin gave an Rh "blocking" titre of 32,000, and the same test cells against the patient's serum diluted with group AB serum gave a titre of 64 to 128. In this respect, therefore, albumin[1] is vastly superior to group AB serum as a diluent.

As a result of this work it has occurred to us that as red cells in the human body are agglutinated and hæmolysed in the presence of Rh "blocking" antibody, causing transfusion reactions or hæmolytic disease of the newborn in maternity cases, and as test cells suspended in serum, plasma or albumin are agglutinated in the presence of "blocking" antibody on slides or in test tubes, while the same test cells suspended in saline solution are not agglutinated when saline solution is used as the diluent for the patient's serum, it may then be possible to explain this simply by postulating that saline solution alters the configuration of the cell surface, whereas in serum, plasma or albumin the cells remain in their normal state, and all (or particular) cell linkages (haptens) are thus readily available for agglutination. We have some support for this theory, in that, if the test cells are suspended in albumin or serum and the "blocking" serum is serially diluted with saline solution instead of serum, then we still obtain strong agglutination, but to a lower titre than when the diluent used is serum or albumin. Also, it would seem that the change in cell structure is reversible, because test cells

[1] Attempts to reproduce these results using a highly purified egg albumen solution instead of human albumin have failed.

TABLE III.

Titrations of Patient's Serum (Mrs. M.H.) with various Diluents against Red Cells in various Suspending Fluids.[1]

Diluent for Serum.	Group of Test Cells.	Suspending Medium for Test Cells.	Final Dilution of Patient's Serum.																
			2	4	8	16	32	64	128	256	512	1,024	2,048	4,096	8,192	16,384	32,768	65,536	131,072	262,144
Normal saline solution.	O MN Rh_1	Normal saline solution	Tr.	—	—															
		Rous-Turner solution ..	1	—	—															
		Group AB serum ..	4[2]	3	3	3	2	2	Tr.	—	—									
		25% albumin	4	4	4	4	3	3	3	3	3	3	2	2	1	1	—	—		
		Washed cells (neat)[3] ..	3	2	1	—	—													
Normal group AB serum.	O MN Rh_1	Normal saline solution..	Tr.	3	2	2	1	Tr.	—	—										
		Rous-Turner solution ..	1	3	3	3	3	2	1	—	—									
		Group AB serum ..	4	4	4	4	4	4	4	4	4	4	4	3	2	1	—	—		
		25% albumin	4	4	4	4	4	4	4	4	4	4	4	4	3	3	3	2	2	1
		Washed cells (neat) ..	4	4	4	4	4	4	4	4	4	4	4	3	3	1	—	—		
25% strength human albumin.	O MN Rh_1	Normal saline solution..	Tr.	4	4	4	4	4	4	4	3	3	3	2	2	2	1	—	—	
		Rous-Turner solution ..	1	4	4	4	4	4	4	4	3	3	3	2	2	2	1	—	—	
		Group AB serum ..	4	4	4	4	4	4	4	4	4	4	4	4	3	3	3	3	2	1
		25% albumin	4	4	4	4	4	4	4	4	3	3	2	2	1	—	—			
		Washed cells (neat) ..	4	4	4	4	4	4	4	3	3	2	1	—	—					

Wiener's Original Test for Rh "Blocking" Antibody.

Normal saline solution.	O M Rh_1	Normal saline solution	—	—	—	—	—	—	—	—	—	—	—	—						
After one hour, known anti-Rh_0 serum added, incubated for one hour, result read again			—	—	—	—	—	—	—	1	2	3	3	3						

[1] This table presents one of a series of protocols; all tests were carried out at 37° C.; this "trial bleed" was obtained sixty-eight days after the transfusion reaction.
[2] The figures 4, 3, 2 and 1 indicate degrees of agglutination.
[3] The use here of washed cells (neat) followed work by Simmons and Christie (unpublished).

washed three times with saline solution when used neat, and added to a "blocking" serum serially diluted with serum or albumin, are also agglutinated strongly and to high titres. The finding that saline solution in the cell-serum or cell-albumin mixtures does not prevent agglutination disagrees with Wiener's theory that the success of the conglutination test depends partly on the absence of any added crystalline solution.

Attempted Re-immunization of the Patient.

As no specific anti-Rh′ serum had previously been found in Australia, and as only a few millilitres of the potent serum had been obtained, we attempted to reimmunize this patient by bi-weekly intravenous injections of 0·2 millilitre of group O Rh′ cells suspended in Rous and Turner solution. The patient was given four injections, but her veins were difficult to locate and inject, and also her general condition precluded further attempts. There was, however, slight evidence of titre increase in blood taken subsequently. Hill *et alii*[13] have successfully used larger doses (up to four millilitres) of cells for reimmunization; but such large doses were not deemed wise in this instance in view of the patient's condition.

Soon after our attempted reimmunization of this patient, the Commonwealth Serum Laboratories received from Dr. W. W. Hallwright, of the Wellington Hospital, New Zealand, a generous supply of a potent anti-Rh′ serum for distribution. The details of his case, one of Rh iso-immunization in pregnancy, will be published in a New Zealand journal early in 1946.[1]

Discussion.

This case and the subsequent investigations demonstrate several important points. The first is the danger of repeated transfusions without previous determination of the Rh type of the patient. This point must be again emphasized. The giving of repeated transfusions to patients whose Rh type is not predetermined will inevitably lead to transfusion reactions, as it did in this case. Whilst this point is important, and it is recommended that every hospital with adequate facilities and trained laboratory personnel should perform this Rh test as a routine measure on the blood of all patients likely to have repeated transfusions, it is not advisable to magnify the problem out of its true perspective.

It should be remembered that the initial reaction in an Rh-negative patient due to repeated transfusions of Rh-positive blood is usually mild, and that the reactions become increasingly severe or even fatal. Therefore, any patient who has a transfusion reaction, however mild, should not be given another transfusion without an investigation from the Rh point of view, or at any rate without an attempt to determine the cause of the reaction. Moreover, it is known that of those subjects who might become sensitized, only one or two in fifty actually do so.

Levine,[13] dealing with unintentional iso-immunization of the Rh-negative female population, has issued a report which should be borne in mind, particularly when more Rh-testing facilities are available. In a vast number of cases studied by him he found 25 Rh-negative women whose first-born suffered from *erythroblastosis fœtalis*, and of these women 16 had a history of previous blood transfusion. There were 10 fœtal deaths in this group of 16. Levine makes the recommendation that no blood transfusion should be given to young women, girls or female infants unless tests for the Rh factor are carried out. Those found to be Rh negative must receive Rh-negative blood. As it is impracticable, without adequate laboratory facilities and trained personnel throughout the country, to determine the Rh type of every patient who is to receive a blood transfusion, it is obvious then that the only practical procedure is to have Rh tests performed when a definite indication exists, particularly in the case of pregnant or parturient women, or in cases in which multiple transfusions are required. When this cannot be done, the recent compatibility tests which are discussed here are now so sensitive and simple that it should be possible to find compatible donors without any transfusion risk to the patient, even if she is already sensitized.

[1] See *The New Zealand Medical Journal*, February, 1946, page 2.

One important technical point which must not be overlooked in connexion with this procedure is that, in a case in which it is known that the patient will require multiple transfusions, it is preferable to determine his Rh type before the first transfusion. This is wise, because, if the patient is Rh negative, the first transfusion of Rh-positive blood may not be destroyed (as the patient is not yet sensitized), so that the Rh-positive (donor) cells will still remain in the patient's circulation. A sample of blood taken from the patient soon after this transfusion would give the type erroneously as (weak) Rh-positive, and so the whole point of possible Rh sensitization might be missed.

The second important point is the accidental transmission of malaria by blood transfusion. The transmission of diseases by blood transfusion has always been one of the problems in a successful service. All precautions can be taken to exclude contaminating organisms, but bacteria or parasites already in the donor blood may present a problem. Obviously no donor who is not in perfect health should be used, nor any donor with a known septic focus. The advisability of testing donors for syphilis is generally known and has been widely discussed, and will not be dealt with here. The problem of malaria transmission in Australia was one which rarely had to be considered prior to the war. We have no tests for latent or inactive malaria. Examination of a blood film (thick or thin) gives negative results more often than not in such cases. It was formerly thought that it was sufficient to exclude donors who had suffered from malaria within, say, the last five or six years. However, the recent report of McClure and Lam[14] disproves this point. These authors reported two cases of malaria resulting from the transfusion of blood which had been stored in the refrigerator (one bottle for five days). The donors were related and had spent their childhood in Sicily. However—and this is important—neither of them had had any symptoms of malaria for twenty-five years. It is advisable, therefore, to exclude donors who have lived in malarious areas, whether they have or have not suffered from malaria. Of course, as McClure and Lam point out, the blood may be converted into plasma, for filtration renders it free of parasites. An official definition of "the malarious area" so far as this country is concerned, is incorporated in a handbook published by the Red Cross Blood Transfusion Service (Western Australian Division) and kindly sent to us by the honorary director, Dr. G. A. Kelsall; the definition given is "New Guinea and the South-West Pacific Islands lying West of 170° E. long. and North of 20° S. lat., with the exception of Thursday Island and Horn Island". "Potentially malarious area" is roughly that part of Australia north of 19° south latitude, except the Atherton plateau above 2,000 feet and the coastal strip up to and including Broome in Western Australia.

Rubenstein *et alii*[15] also report 12 cases of post-transfusion malaria. In five of these cases from twelve to twenty-seven years had elapsed since the donors resided in a malarious area, and moreover, not one of these donors had a past history of manifest malaria. It is of interest that in nine of these twelve cases the infecting parasite was the *Plasmodium malariæ* (quartan). Whilst post-transfusion malaria is rarely fatal if quickly recognized and treated, it is an added burden for a patient already taxed enough to need a blood transfusion, and naturally should be prevented whenever possible. The "remedy" is the rejection of all donors who have lived in malarious areas. Whether or not their blood is stored, whether or not they have had manifest malaria, and whether or not (in the case of service personnel) they have had prophylactic "Atebrin" or other anti-malarials, they must be regarded as potential transmitters of malaria.

In the present case the donor should obviously have not been used, as he had known recent malaria. Two other cases of post-transfusion malaria are known to one of us to have occurred in Australia in recent years, and there have probably been others. We know that this danger is being carefully watched by the various divisions of the Red Cross Blood Transfusion Service in Australia, but it is timely that the warning should be sounded again.

The third important point is the production of a specific anti-Rh′ (70%) antiserum (the first reported in Australia) by iso-immunization of an Rh-negative subject by Rh-positive donors of sub-type Rh_1.

As was mentioned in the quotation from Wiener,[11] the Rh_0 antigen is by far the most antigenic. Here we have an Rh-negative patient immunized by the Rh_1 (Rh_0′) antigens (the last four donors were of Rh_0′ type), who yet at first sight seemingly produced an anti-Rh′ antibody to the exclusion of the anti-Rh_0 antibody. This would be in complete disagreement with the statement that the Rh_0 antigen is by far the most antigenic, Rh′ falling well behind in antigenicity. The matter was elucidated, however, by an investigation of the "blocking" properties of the serum. As will be seen from Table III, the serum contains a potent anti-Rh_0 "blocking" antibody, which by Wiener's blocking test was shown to have a titre of 512, but which by the conglutination or albumin test had a titre of at least 8,000 to 16,000. (These tests, part of a series, were performed on a sample of the patient's serum collected about ten weeks after the transfusion reaction, at which time the anti-Rh′ agglutinating titre estimated by the ordinary saline method had dropped from 512 to two.) Thus the antiserum produced was actually an anti-Rh_0′ serum; in it the anti-Rh_0 "blocking" antibody had neutralized the anti-Rh_0 agglutinating antibody if this was originally present. In other words, the serum produced was a "specific" but not "pure" anti-Rh′ serum, and the "blocking" antibody had converted an anti-Rh_0′ (87%) serum into an anti-Rh′ (70%) serum. The same thing can be done *in vitro* to obtain specific anti-Rh′ and anti-Rh″ (30%) serum for Rh typing from the corresponding polyvalent antiserum. The production of a "pure" anti-Rh′ serum is an extremely rare occurrence; but it can take place when an Rh_2 (Rh_0″) person is immunized (by a fœtus or blood transfusion) by an Rh_1 (Rh_0′) or Rh_1Rh_2 (Rh_0′Rh_0″) person. Since both the patient's blood and the donor or fœtus's blood contain a common Rh_0 antigen, the patient can react only to the Rh′ antigen. Similarly an Rh_1 person can produce a "pure" anti-Rh″ serum by immunization with blood of Rh types Rh_2 or Rh_1Rh_2. This has occurred in a few cases (all through pregnancy), as described by Levine and Waller[12] and Wiener *et alii*,[13] and these cases yielded antisera which, of course, are ideal for Rh typing (as distinct from simple Rh testing). However, although the necessary matings are quite common, these "pure" sera are but rarely produced, because of the poor antigenicity of the Rh′ and Rh″ antigens. To date no sample of serum of specificity anti-Rh_0″ or anti-Rh″ has been found in Australia, although a constant search is in progress.[1]

The fourth point is the value of the Rh "blocking" test as a guide in preventing transfusion reactions. The value of Wiener's "blocking" test is graphically demonstrated in Table III. This specimen of serum, obtained sixty-eight days after the last transfusion of Rh-positive blood, when tested by the usual tube or slide technique of saline dilutions and with Rh_1 cells suspended in saline solution, had a weak Rh-agglutinating titre of two; this might be missed in an ordinary laboratory compatibility test, particularly if it was carried out at room temperature. If, now, to the same set of tubes we add two drops of an anti-Rh_0 agglutinating serum (suitably diluted), mix and reincubate and read the result again, we find that the agglutination of the cells[2] is "blocked" out to a dilution of approximately 512, and thus it is proved that a potent Rh "blocking" antibody is present.

As this "blocking" antibody is just as lethal as the usual Rh agglutinating antibody to transfused incompatible red cells, if not more so, the importance of the test for blocking is apparent. It is possible for sensitization (either by transfusion or by pregnancy) to occur, in which the only antibody present is the "blocking" type. This explains some of the cases in which, although obvious sensitization to the Rh factor has taken place, there is no *in vitro* agglutination of test cells. Such cases can be very puzzling if advantage is not taken of the recent tests for Rh "blocking" antibody.[1] These "blocking" antibodies are common as the result of iso-immunization in pregnancy, but until recently no actual case had been reported of a transfusion immunization resulting in the production of "blocking" antibodies without agglutinating antibodies. However, Unger and Wiener[20] have recently given details of such a case, in which a "blocking" antibody titre of 480 was present and which caused, and was detected following, a transfusion reaction. Wiener's original "blocking" test has now been superseded by the conglutination and albumin tests.

The fifth important point is the greater value of the conglutination test (compatible serum being used for cell suspensions) or the albumin test, 20% to 25% strength serum albumin being used as a suspending medium for red cells in tests for Rh "blocking" and agglutinating antibodies, or in cross-compatibility tests.

The sixth point is the higher Rh "blocking" titres which are found when a combination of the conglutination and albumin tests is employed for Rh titre determinations.

The conglutination test (test cells suspended in normal group AB serum and patient's serum serially diluted with group AB serum), or the more recent substitution of 20% to 25% strength albumin as the suspending fluid or diluent instead of serum, has a greater value than the blocking test, in that both these tests give pronounced agglutination with both Rh "blocking" and agglutinating antibodies in a direct one-stage test. Table III shows a convincing demonstration of the superiority of the last-mentioned tests. Here is shown the patient's serum, which, when tested with cells in saline solution *versus* saline dilutions of the test serum (original Rh-agglutination test), gives an Rh-agglutinating titre of only two; this, as was previously mentioned, could be missed. Wiener's blocking test shows the serum to have an Rh "blocking" titre of approximately 512, but by the conglutination test and by the albumin test the titre was 8,000 to 16,000. The original saline test is a true measure of the ordinary Rh-agglutinating antibody titre,[23] which we have confirmed with a number of specimens of anti-Rh serum; but the serum or albumin tests measure both agglutinating and blocking antibody titres. However, when a combination of the conglutination and albumin tests was used, the Rh "blocking" titre was found to be 262,000. One could quite well imagine the severe transfusion reaction or severe fœtal damage which could occur with such a high-titre Rh "blocking" serum as that mentioned above, and yet the presence of Rh antibodies might have been missed in the past by the usual cross-matching tests prior to transfusion or in saline titre determinations.

Routine Rh Testing Prior to Transfusion.

Another point worthy of mention is that, while the standard anti-Rh_0 agglutinating serum will generally be sufficient in the small laboratory for clinical Rh testing, amongst those subjects classed as Rh-negative will be some of the rare types, Rh′ (1%), Rh″, Rh′Rh″ and Rh_y (very rare). In a properly organized donor service these types will be eliminated by the use of the rarer Rh sub-type sera anti-Rh′ and anti-Rh″. Some persons known to be Hr negative will also be listed if possible, should such donors be required.

In the case described by us we have a true Rh-negative patient (genotype rhrh) who had been sensitized by the Rh_1 antigen and had developed anti-Rh_0 (blocking) and anti-Rh′ (agglutinating) antibodies. If the subsequent Rh-negative donors used were chosen by the use of anti-Rh_0 serum only, an Rh′ individual would be regarded as Rh negative and hence seem a suitable donor. Cross-matching tests would show up the incompatibility; but if by any chance these tests were omitted, the patient would suffer a

[1] Since we prepared this paper for publication, a specific anti-Rh″ serum has been found by the Red Cross Blood Transfusion Service (New South Wales Division).

[2] Actually the "blocking" titre in Table III was determined with group $OMRh_2$ cells, as they do not produce agglutination with an anti-Rh′ antibody. As the titre of the anti-Rh′ agglutinating antibody was now only two, we could have used Rh_1 cells equally well at this stage.

[1] The details of another more intricate test by means of rabbit anti-human-globulin serum has recently been described (see Coombs, Mourant and Race, *The British Journal of Experimental Pathology*, Volume XXVI, August, 1945, page 255).

severe, if not fatal, hæmolytic reaction. Cappell[22] has pointed this out recently, and suggests classifying blood (from a practical viewpoint) into an Rh-positive type if it contains the Rh_0 antigen or an Rh-negative type if it lacks this antigen. As he remarks: "The possession of the dominant antigen Rh_0 is the fundamental thing which should determine whether an individual requiring transfusion is called Rh positive or Rh negative." From the therapeutic standpoint the person who lacks the Rh_0 antigen should receive transfusions only of Rh-negative blood. The donor, however, must be truly Rh-negative (that is, of genotype rhrh).

If we consider, from the point of view of blood transfusion, the extremely rare type of cases mentioned by Diamond and Denton,[23] in which blocking antibodies of both Rh_0 and Rh' specificity were encountered, cross-matching tests with an Rh' donor (believed to be Rh negative if only anti-Rh_0 serum had been used for testing) by the usual means would obviously reveal no agglutination; but the blood is actually incompatible and would be destroyed *in vivo* with subsequent reaction.

Here again we see the superiority of the conglutination test of Wiener or the albumin test of Diamond and Denton in detecting Rh sensitization. In any of these cases just mentioned, direct incompatibility would be shown by the use of either of these tests.

Summary.

1. A case is described of an Rh-negative patient who, as a result of blood transfusions, was not only sensitized to the Rh factor but also infected with malaria.

2. The antibodies found in the patient's serum as the result of Rh iso-immunization have been analysed.

3. The measures necessary to avoid these preventible accidents are described and the value of the various tests to detect Rh sensitization are discussed.

4. Recent tests made by us in addition to those reported here show that cells suspended in 25% strength human albumin are more sensitive than cells suspended in normal group AB serum when tests are made for the presence of Rh agglutinating or "blocking" antibodies; this confirms the claim of Diamond and Denton that albumin is the most satisfactory suspending medium.

5. For the determination of the Rh "blocking" titre a combination of the two tests (conglutination and albumin) gives the highest titre readings. The testing of cells in albumin against the patient's serum serially diluted with normal group AB serum gives excellent results.

6. Cells suspended in 20% to 25% strength albumin or, in its absence, in compatible human serum, should now be used in all cross-compatibility tests prior to transfusion. The use of saline solution as a suspending medium for the test cells should be strictly avoided. The tests should be performed at both 37° C. and at room temperature.

7. A simple theory is advanced to explain why cells suspended in albumin or serum are agglutinated by Rh "blocking" antibody and why cells in saline solution fail to be agglutinated.

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THE RESULTS OF PENICILLIN THERAPY IN THE TREATMENT OF GONORRHŒA.[1]

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Sydney.

IT is difficult for a junior member of the medical profession to talk with authority on any subject from personal experience. There is open to the resident medical officers in hospitals, however, a fund of information to be obtained from hospital records, not the least of which is how not to compile histories. The resident medical officers in

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on November 29, 1945.

hospitals, more than any other group of medical practitioners, have golden opportunities for research into hospital records.

The subject of venereal disease is one of intense interest and great national importance. Just how important it is can be seen by examining the age incidence of all venereal disease among females in the past four years, which are the war years (see Table I). The effect of venereal disease

TABLE I.
Age Incidence for all Venereal Disease in Females for the War Years.[1]

Year.	Age Group.		Total Incidence Under 25 Years.
	16 to 20 Years.	21 to 25 Years.	
1941	18·3%	24·1%	42·4%
1942	22·9%	25·8%	48·7%
1943	27·9%	27·9%	58·8%
1944	23·0%	28·9%	51·9%

[1] Of total number of patients, 52·7% were married.

and of the social and psychological conditions associated with the actual infection is a grave threat to our future mothers and to our national family life. Over the past four years, the incidence of venereal disease occurring in young girls and women aged under twenty-five years rose from 42·4% in 1941 to 58·8% in 1943 and 51·9% in 1944. From the point of view of the future fertility of these patients, it is in this age group that the most tragic damage is done. Of the total, 52·7% were married women. These young women come from home conditions of frustration and careless, hasty marriages. The effect of the war stresses and the influx of Allied servicemen with lonely hearts and bulging pockets have caused the figures for venereal disease to soar.

This paper will deal only with gonorrhœa, as the treatment of this disease has varied so dramatically over the past few years. During the war years, with the rise in the incidence of venereal disease, the figures for gonorrhœa are interesting (see Table II). The dramatic figure for

TABLE II.
New South Wales Incidence of Gonorrhœa.

Year.	Males.	Females.
1941	2,936	465
1942	3,857	615
1943	2,603	819
1944	2,106	1,205
To October 31, 1945	2,213	595

1944 is due not only to the influx of Allied servicemen, but also to the careful tracking down of suspected sources of infection by the American army authorities, and the subsequent fall is largely due to removal of this source of infective patients. For example, in 1944, 2,280 notifications of possible sources of infection were received by the Board of Health; 1,423 of these were received from the American army, 851 from other sources. Of these notified sources, 835 were located, and 486 of the subjects or 58·2% were found to be infected; this means, if the same ratio is used, that about 800 possibly infected people were never found.

In addition to voluntary patients and patients notified as sources of infection, the National Security Regulations, in force during the war, brought another group of patients to venereal disease clinics—vagrants who were ordered by court to attend a clinic for examination. In 1944, 352 women vagrants were directed to attend for examination. Of these, 183 attended and 96 or 52·4% were found to be infected.

THE RACHEL FORSTER HOSPITAL SPECIAL CLINIC.

In assessing the results of penicillin therapy in gonorrhœa, I have examined the records of the Rachel Forster Hospital, Redfern, over a period of fifteen years, from 1930 to the end of October, 1945. During this period the attendances of new patients have remained fairly constant until 1937, with a rise prior to and during the first war years, and the tremendous rise in the last three years is due partly to the arrival of Allied servicemen in Australia and partly to the factors mentioned above (see Table III). This clinic is the largest venereal diseases clinic for women in Australia, and has been functioning since 1926 so successfully that the hospital has had a hard struggle to be recognized as a general hospital and is still sometimes regarded as a venereal diseases hospital only.

These results therefore apply only to female patients, but I shall make some comparisons with treatment of male patients.

Corresponding to this fifteen-year period, I have examined 3,563 gonorrhœa histories (see Table IV). The results are compiled from the records of 2,434 patients who completed their treatment at the clinic. At first glance one

TABLE IV.
Patients Examined in Each Group.

Treatment.[1]	Patients Completing Treatment.	Patients Transferred Elsewhere.	Defaulters.	Percentage of Defaulters.
Local	1,054	336	736	22·8
Sulphonamide ..	1,093			
Penicillin	287	8	49	14·3

[1] The number of patients examined was 3,563; the number who completed treatment was 2,434.

of the results of penicillin treatment is seen to be a fall in the percentage of patients who defaulted from treatment before cure. Of all patients treated before the introduction of penicillin, 22·8% defaulted, whereas in the penicillin group 14·3% defaulted. This, as will be seen later, is probably related to the shorter time and more convenient methods of treatment.

The patients are divided into three groups—those treated (i) by local methods only, (ii) by sulphonamides and (iii) by penicillin.

Diagnosis.

The diagnosis rests on the demonstration of the Gram-negative intracellular diplococci in smears taken from infected parts. Extracellular diplococci resembling gonococci in shape and staining reactions are regarded as suspicious and worthy of further investigation if clinical findings and history suggest the possibility of infection. Cultural examination of the discharge is satisfactory only if carried out with a special selective technique.

Positive Reaction to the Gonococcal Complement Fixation Test.

The question now arises of the significance of the positive gonococcal reaction complement fixation test for

TABLE III.
New Attendances, Rachel Forster Hospital Special Clinic.

1931	1932	1933	1934	1935	1936	1937	1938	1939	1940	1941	1942	1943	1944	1945
236	261	247	257	219	263	326	303	405	365	330	322	1,269	1,312	1,011

gonorrhœa (see Table V). The positive gonococcal reaction is not regarded as diagnostic of gonorrhœa. It is variously regarded as being an indication of a gonorrhœal focus somewhere in the body, and as an indication of previous infection. It is probable that there are many fallacies for this test—for example, *Micrococcus catarrhalis*. The

TABLE V.
Gonococcal Reaction of 789 Non-Notifiable Patients.

Test.	Number.
Result of test:	
Positive gonococcal reaction ..	574 cases.
Incomplete positive gonococcal reactions	215 cases.
Second blood test	334 cases.
Number of tests made:	
One	200 patients.
Two	230 patients.
Three	231 patients.
Four	85 patients.
Five or more	43 patients.
Total with three tests or more ..	359 patients.

explanation for negative results in patients from whom smears have been examined with positive findings, has been that in these cases there is free drainage of infective material.

I do not wish to enter into the controversy over the cause of a positive gonococcal reaction, but to quote some figures from the clinic (see Table V). In a series of 789 patients who attended the clinic for examination, and who were never proved to be infected, a positive or incomplete reaction was found in the first or subsequent blood test. It is the routine of the clinic whenever possible and if the patients will attend—for they cannot be compelled to do so—that any patients with a history or clinical findings suggestive of a possible Neisserian infection, should have in addition to the smear examined on the first attendance, two subsequent smears examined as soon as possible after the following menstrual periods, and a second blood test at an interval of two months, before the patient is reassured that she is free from infection. Any concealed syphilitic infection is found at the second blood test, but as it is for this reason that the second blood sample is taken, the gonococcal reaction is not always recorded, nor is this test always requested. In this series of 789 non-notifiable patients a second blood test was made in 334 instances; this means that in some 400 odd cases a positive Wassermann reaction may have been developing. Four hundred and thirty did not have the required number of confirmatory smears, and in spite of the positive gonococcal reaction could not be notified as giving positive results or forced to attend for further tests. Three hundred and fifty-nine patients who had three or more smears examined were considered free from infection.

Of these non-notifiable patients, 126 or 15·9% had associated conditions which would give support to the possibility of previous infection, as outlined in Table VI. Sulphonamides had previously been taken with or without medical advice by fourteen patients.

Positive Gonococcal Reaction in Infected Patients.—Of 1,830 infected patients whose gonococcal reaction was recorded at the first blood examination, 1,204 or 65·8% gave

TABLE VI.
Associated Conditions in Non-Notifiable Patients with Positive Gonococcal Reaction.

Condition.	Number of Patients.
Syphilis	64
Salpingitis	4
Previous gonorrhœa	13
Bartholin's abscess	2
Trichomonas vaginalis	32
Monilia	7
Venereal warts	4

a positive gonococcal reaction, and 626 or 34·2% gave no gonococcal reaction (see Table VII). Of these, 554 or 30·3% continued to give positive reactions in spite of treatment, 342 or 18·7% ceased to give reactions after treatment, 280 or 15·8% continued to give no reactions in spite of infection, and 164 or 9%, who gave negative results at the first examination, gave positive results at the second.

TABLE VII.
Gonococcal Reactions among 1,830 Confirmed Gonorrhœa Patients.

Examination.	Positive Gonococcal Reaction.	No Gonococcal Reaction.	Unrecorded.
First examination ..	1,204 (65·8%)	626 (34·2%)	—
Second examination:			
Previous positive result	554 (30·3%)	342 (18·7%)	298 (16·3%)
Previous negative result	164 (9·0%)	280 (15·8%)	192 (9·4%)

In the total number of treated patients, 159 yielded "positive" smears only after two or more tests; this indicates the need for repeated tests if infection is suspected. The positive gonococcal reaction can only be regarded as an indication for further tests, and even if no gonococcal reaction is produced, repeated smears should be examined if the history or clinical findings are suspicious, and cervix, urethra and rectum should all be examined.

Criteria of Cure.

The persistence of a positive gonococcal reaction does not mean that the patient is not cured. At the clinic, patients are discharged after negative results have been obtained on the examination of three smears, and if the patient is clinically free from infection. Persistence of a discharge may be due to a concurrent trichomonas or monilia infection. A second blood test is always made as a routine measure to detect possible early syphilis, which may have been missed in the earlier test. In the case of female patients this means a second test after an interval of two or three months; but among males, as the duration of treatment has now been reduced to about six weeks, it is possible that a positive Wassermann reaction may develop after the patient's discharge from the clinic. If possible all patients should have the second test at an interval of three months or more after the first.

In males, smears are taken after prostatic massage, at intervals of one or two weeks, and when the result is negative, a sound is passed and examination of smears is repeated. The patient is discharged from the clinic after three consecutive smear examinations have given negative results.

Details of Treatment.

Local Treatment.

I have grouped all the methods of treatment used before the introduction of sulphonamides under the heading of local treatment. Briefly, the patients attended the clinic at varying intervals, from each day to each week, for vaginal douches, application of various antiseptics to the cervix, urethral dilatation and massage and urethral and rectal irrigations, according to the clinical signs. Included in this group are patients treated with various vaccines and by protein shock, none of which appeared to alter the course of the infection to any great extent. Tests were made only when clinical signs were improving. Rectal examinations were not carried out as extensively as now, unless there were signs of infection, such as rectal hæmorrhage and pain. Children were treated in addition with injections of theelin. Complications such as Bartholin's abscess, erosions and salpingitis were treated symptomatically as they appeared.

Sulphonamide Treatment.

With the introduction of the sulphonamides, treatment changed vastly. The attendances were reduced to once or twice a week, unless special indications were present. Urethral and rectal irrigations were continued. The drugs

were given sometimes as one course of about fifteen grammes, sometimes as two consecutive courses of one or two types of sulphonamide, according mainly to the appearance of the patient and the opinion of the physician. Further courses were given as necessary, if clinical appearances failed to improve and if smears remained "positive". Local application of a sulphonamide was tried in resistant cases, and before penicillin was introduced sulphaguanidine *per rectum* was tried for rectal infections, but in this clinic the cases were too few for the value of the results to be assessed.

Penicillin Treatment.

The routine form of penicillin treatment in use at the clinic is as follows. As soon as possible after the diagnosis is made, the patient is admitted to hospital for one day, and is given a course of 100,000 units of penicillin, 20,000 units intramuscularly every three hours for five doses. Children are given the same dose as adults. Forty-eight hours after the completion of the course, or as soon as practicable, the patient reports back to the clinic for smears to be taken. If these are "positive", she is admitted to hospital for more penicillin treatment; if they are "negative", she reports back to the clinic every week for observation and local treatment. Smear examinations are repeated after each menstrual period.

Local treatment consists of the following procedures: (i) swabbing of the vagina with "Dettol" and glycerin; (ii) application of "Titrol" to the cervix; (iii) application of argyrol to the urethra; (iv) dusting with "Dermatol" powder. This weekly treatment enables any complication such as Bartholin's abscess to be treated, and keeps the patient aware that she is still not cured. Cervical erosions are painted with a 40% solution of silver nitrate at intervals of two, three or four weeks, according to the response.

The rectum is examined and smears are taken at the first visit, and if these yield positive findings, smear examinations are repeated when the post-menstrual smears are examined. In any case, the rectum is examined when smears are taken. No local rectal treatment is given, and no local treatment to the vagina or rectum is advised at home.

If at any time the smear findings become positive, the patient is readmitted to hospital for a further course of penicillin treatment. After two courses of penicillin treatment, if the patient still yields positive findings and appears to have a true resistance to infection, a course of 200,000 units is given. When admission to hospital is delayed, treatment with sulphadiazine is commenced in the meantime. Patients who develop salpingitis are admitted to hospital and treated with sulphadiazine with or without further penicillin, according to the case. Sulphaguanidine is still used for some of the persistent rectal infections.

Male patients at the Board of Health Clinic are treated as out-patients and are given a course of 150,000 units of penicillin—30,000 units intramuscularly every two hours for five doses. This means that while female patients have a maximum concentration of penicillin lasting over about fifteen hours, male patients have a maximum concentration lasting only ten hours, which is compensated for by the higher dose used.

When penicillin was first used, weekly attendances were abandoned, and patients attended at the clinic only for the examination of post-menstrual smears. Although not many patients were treated in this way, it was considered that too many required second courses of penicillin (see Table VIIIa). Of 25 patients treated with penicillin alone, four required two courses of penicillin. In the total series, 28 out of 287 required more than one course. This method has now been replaced by weekly attendances. Altogether 257 patients gave negative results to tests at the first testing, and continued to do so, two patients who later gave positive results were treated with sulphonamides, and 28 required more than one course of penicillin. The results of tests became positive up to intervals of three months (Table VIIIb); this indicates the need for observation for at least three post-menstrual smears. Many of the patients who gave positive results after one or two negative results admitted intercourse during treatment, and probably many other relapses were due to the same cause.

TABLE VIIIa.
Courses of Penicillin Treatment Required (287 Cases).

Number of Courses.	Total Number of Cases.	No Local Treatment.
One	259	21
Two	22	4
Three	6	—
Percentage having more than one course ..	9·7	16

TABLE VIIIb.
Analysis of Positive Results in Smears after Penicillin Treatment.

Time of Positive Findings.	Number of Cases.
Two to four days	5
First post-menstrual smear	14
Second post-menstrual smear	16
Third post-menstrual smear	4

We now come to an analysis of the results of penicillin treatment, as compared with the other methods of treatment. Table IX indicates the effect of penicillin on the time taken to produce the first permanent "negative" smear. The forty-eight hour results are placed for comparison in the "up to two weeks" column. The figures in parentheses denote some cases in which treatment was instituted on a positive result obtained elsewhere without a confirmatory test made at the Rachel Forster Hospital before treatment, so that the first test made here gave negative results. Now that all smears are examined at the hospital, all patients have one or more confirmatory smears examined at the clinic before penicillin is given. The analysis is made in months to facilitate comparison. In the "local" group, the cases are spread out up to the fifth month with a peak at two months. One patient, who attended regularly, took over two years for her smears to become "negative"; during which time she had vaccines and protein shock therapy in addition to the other extensive treatment. In several cases in which local treatment had

TABLE IX.
Duration of Treatment up to First Permanent "Negative" Smear.

Method of Treatment.	Period of Treatment.										
	Up to Two Weeks.	One Month.	Two Months.	Three Months.	Four Months.	Five Months.	Six Months.	Six to Twelve Months.	One to Two Years.	Over Two Years.	Total.
Local	86 (23)	124	205	193	157	70	56	139	23	1	1,054
Sulphonamide	258 (20)	357	209	113	54	28	23	44	7	0	1,093
Penicillin	253	10	8	7	7	1	1	0	0	0	287

TABLE X.
Duration of Attendance After Commencement of Treatment.

Method of Treatment.	Period of Treatment.							
	Up to Two Months.	Three Months.	Four Months.	Five Months.	Six Months.	Six to Twelve Months.	One to Two Years.	More than Two Years.
Local	4	24	62	85	112	569	185	12
Sulphonamide	49	251	247	190	128	208	20	0
Penicillin	14	134	70	33	15	21	0	0

been given for several months without improvement and then sulphonamide used, the condition cleared up as in any other case in which the sulphonamides were used. In the sulphonamide-treated group, smears from more than two-thirds of the patients had become "negative" in two months, and only a small proportion required more than twelve months' treatment. Once again, some of the patients resistant to the sulphonamides responded easily to penicillin. In the penicillin-treated group, about 80% of the patients gave negative results in less than two weeks. Resistant patients are considered to be those requiring more than two courses of penicillin, and so far no patients have required more than three courses of penicillin (see Table IX).

Duration of Treatment.

The duration of treatment for the different methods is compared in Table X. In each case, when sulphonamide or penicillin was used for a patient previously treated by another method and still infective, the calculations are made from the time of commencement of the new method. Whereas in the "local" group three-quarters of the patients attended for more than six months, and many for more than one year, the greatest number are now discharged from the clinic in less than four months; in actual fact the longest attendance was nine months, by a patient who attended irregularly. The group discharged in less than two months is made up of children, of women who had more frequent menstrual periods, and women who were not menstruating either owing to pregnancy or from some other cause, and upon whom tests were made at more frequent intervals after provocative treatment to the cervix and urethra. In the case of pregnant women it is often desired to complete treatment before confinement, and these patients are sometimes given a 200,000 unit course followed by more frequent tests.

The "local" group also covers the period of the financial depression, when many of the women attending were destitute and living only on food relief. Better living conditions and higher wages during the war may be an important contributory factor to an increased natural resistance in the "penicillin" group.

Incidence of Relapse.

In examining the incidence of relapse, I have considered the cases in which the results of tests became positive after having previously been negative, and not the actual number of relapses, which could be compared only under very ordered conditions—as, for instance, in the "penicillin" group, in which tests are made after every menstrual period. In the "local" group, tests were made only when some improvement was noted, and results may have been negative several times if the tests were made earlier. When comparisons are made in this way (see Table XI), it can be seen that in the "penicillin" group the patient is much less likely to relapse. Since the main reasons for relapse are, in addition to a possible true resistance to treatment, a further exposure to infection or flare-up by continued coitus, the presence of a chronic deep-seated focus, or failure of any method actually to reach the area of infection, it is obvious that the method of administration of penicillin, and its effects in reducing the time taken to complete treatment, will naturally reduce the incidence of relapse.

TABLE XI.
Incidence of Relapse following Treatment.

Method of Treatment.	Number of Relapses.	Relapse Rate.
Local	137	13·0%
Sulphonamide	138	12·6%
Penicillin	30	10·4%

Incidence of Complications.

In Table XII I have compared the complications manifested during treatment. I have endeavoured to omit any complications which were in evidence before treatment, unless they flared up during treatment, as most of them did. One of the most striking comparisons is the high rate of pelvic inflammation in the "penicillin" group. Among the cases of pelvic inflammation are included salpingitis, pelvic abscess, pelvic peritonitis and menorrhagia. The patients treated with penicillin mainly show menorrhagia and mild salpingitis; but as they are under observation for a much shorter period of time than the others, it is difficult to estimate the amount of permanent damage that is done.

The high incidence of rectal infection in the "sulphonamide" and "penicillin" groups, which is probably more a manifestation of infection than a complication, is most likely due to the increased attention paid to this form of infection, rather than to an actual increase in occurrence. In a proportion rectal smears give positive findings even when no positive results can be obtained from the urethra or cervix (see Table XIII). Some of these rectal infections were apparently truly resistant to sulphonamides. Of all the patients resistant to sulphonamides, sixteen had a persistence of rectal infection only, after smears from the

TABLE XII.
Incidence of Complications During Treatment.

Method of Treatment.	Complication.							
	Pelvic Infection.	Rectal Infection.	Bartholin's Abscess.	Arthritis.	Venereal Warts.	Urethral and Bladder Disorders.	Perianal Abscess.	Recto-vaginal Fistula.
Local	135 (12·8%)	104 (9·9%)	83	7	10	18	6	1
Sulphonamide	95 (8·7%)	275 (25·1%)	80	12	19	6	0	0
Penicillin	32 (11·5%)	55 (19·1%)	5	2	2	0	1	0

TABLE XIII.
Cases of Rectal Infection.

Method of Treatment.	Total with Positive Rectal Findings.	Positive Rectal Findings Only.	Total Relapses.	Relapses Due to Rectal Infection.	Cases of Resistance.	Resistance Due to Rectal Infection.
Local	104	1	137	20	—	—
Sulphonamide	275	10	138	34	55	16
Penicillin	55	7	30	4	28	4

urethra and cervix had yielded negative findings, and of 28 patients resistant to penicillin, four had persistent rectal infections. I have considered sulphonamide-resistant patients to be those requiring more than three courses of sulphonamides, and penicillin-resistant patients to be those who required more than one course of penicillin. In spite of the cessation of local rectal treatment, the incidence of persistent positive findings in rectal smears is lower in the "penicillin" group.

The troublesome bladder and urethral complications, such as cystitis, skenitis and urethral stricture, no longer present the difficulty they did and have disappeared as complications in the "penicillin" group.

Associated Conditions.

Table XIV indicates the incidence of other conditions occurring with the Neisserian infection. The high incidence of *trichomonas vaginitis* with monilia infections in the "sulphonamide" and "penicillin" groups is probably due to the closer attention paid to any discharge persisting after treatment has been instituted. These infections are treated at once, and the attendance at the clinic is usually not greatly prolonged. Trichomonas infections are not regarded as true venereal infections; but there has lately been considered to be some connexion between infections in males and females.[5]

The association of chancres with a Neisserian infection brings up an important question with regard to the use of penicillin in the treatment of early syphilis. To what extent is the administration of penicillin to patients suffering from gonorrhœa likely to mask the appearances of early syphilis? In the "sulphonamide" group one patient developed a chancre at five weeks; all the others were present at the time of examination. The patients all have a smear examined for spirochætes if the appearances are at all suspicious. The dose of penicillin is by no means adequate to cure syphilis; the danger lies in the fact that it may be enough to mask the early chancre or delay its appearance. At the clinic we have had no cases of delayed appearance of the chancre; but at the Board of Health Clinic, one patient developed a chancre at sixty days, which was considered to be a delayed reaction due to the inhibiting action of penicillin, and not due to a reinfection. The second blood test will prevent any such infection from being missed, provided enough time has elapsed to allow the Wassermann test to produce a positive reaction, and in this respect women are better protected than men, owing to the longer observation period.

The following is the typical clinical history of a sulphonamide-resistant patient who responded to penicillin.

Patient D.H., aged forty-three years, first attended the clinic on October 22, 1943, with an infection of probably one month's duration. The Wassermann test and the gonococcal complement fixation test both produced negative results, urethral and cervical smears yielded positive findings, and a rectal smear gave negative findings. Treatment was commenced with one course of "M & B 693", followed by one course of sulphanilamide, and she attended the clinic twice a week for local treatment. Smears taken on November 9, 1943, and November 30, 1943, gave positive findings. On December 14, 1943, she was given one course of "M & B 760" and a course of weekly "Gonoderm" injections (vaccine) was commenced; altogether eleven injections were given. On January 24, 1944, rectal smears gave positive findings, and a second course of "M & B 760" was commenced. On February 1, 1944, urethral and cervical smears gave positive findings, and on February 15, 1944, a second course of sulphanilamide was given. On February 24, 1944, and March 23, 1944, rectal smears only still gave positive results. "M & B 693" local treatment to the rectum was commenced, one tablet being inserted morning and night. On April 18, 1944, a rectal smear only still gave positive findings, and the number of tablets was increased to two morning and night. On July 5, 1944, the rectal smear only still gave positive findings after nine months. One course of penicillin treatment was given on July 8, 1944. On July 10, 1944, examination of post-menstrual smears gave negative results. The next smears on August 8, 1944, all gave negative findings, and the patient then defaulted and was not examined again for five months; but no further "positive" smears were obtained.

The Future of Penicillin in the Treatment of Gonorrhœa.

Penicillin has truly ushered in a new era in the treatment of gonorrhœa. We must now consider what ill effects may arise from its use.

Apart from the flare-up of pelvic symptoms after penicillin treatment, there appear to be as yet no toxic results or ill effects arising out of the administration of penicillin. The question of its possible effect on early syphilis has been briefly discussed.

The main ill effects of penicillin would appear to come from its abuse. Penicillin may produce clinical improvement and a false sense of security in patients still infective, unless they are carefully controlled. The routine examinations are most essential. The need for regular examination of smears brings up the question which was recently raised in a daily newspaper: Why do results of pathological examinations take so long to return from the Board of Health? The answer to this probably lies in the proposals of the Section of Pathology and Bacteriology of the New South Wales Branch of the British Medical Association, outlined in a recent issue of THE MEDICAL JOURNAL OF AUSTRALIA—a State-wide pathology service.[6]

We have seen that the incidence of defaulters has fallen in the "penicillin" group. Most patients accept readily the fact that, although their smears are "negative", they must still return for tests and treatment. There does exist a black market in sulphonamides, and there is a similar possibility in the case of penicillin.

The social danger of penicillin is the result of the removal of the fear of infection, if this does exist to any great extent. We have still to see whether the ease of

TABLE XIV.
Conditions Associated with Gonorrhœal Infections.

Method of Treatment.	Total Cases of Syphilis.	Chancre.	Total Cases of Pregnancy.	Miscarriages.	*Trichomonas Vaginalis* Infections.	Monilia Infections.
Local	52	7	28	4	8	5
Sulphonamide	38	7	30	10	126	36
Penicillin	4	1	8	4	39	4

cure produces an increased disregard for the risk of infection. The problem of gonorrhœa is not only purely medical, but also social, and medical practitioners must play as important a part in the one field as in the other. With us lies a great power for instruction and rehabilitation of the girls in our midst who are unfortunate. In this post-war era we must not make the same mistakes as last time. Treatment has advanced tremendously since then, but much of our legislation remains unaltered. The National Security Regulations are only temporary, and reforms are needed in our venereal diseases acts. The medical profession has the necessary knowledge and experience to cope with these problems.

Briefly the results of penicillin therapy can be summarized thus: (i) reduction in the frequency of attendances necessary and more convenient methods of treatment; (ii) diminution of the period of infectivity; (iii) reduction of the period of attendance at the clinic before discharge: (iv) reduction in the rates of relapse and default from treatment; (v) reduction in the incidence of disabling complications, except for a doubtful effect on pelvic complications.

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THE REHABILITATION OF WAR NEUROTICS.[1]

By C. M. McCARTHY,
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"THIS Committee is concerned with the urgency and extent of the problem of mentally handicapped discharged personnel." This is the text of a resolution adopted by representatives of the Returned Sailors, Soldiers and Airmen's Imperial League of Australia, of the Trades and Labour Council and of the New South Wales Branch of the British Medical Association.

Rehabilitation in general is a political and social problem. The rehabilitation of war neurotics is a special problem of social and medical importance. It is time that, as an organized profession, we set about discussing it and finding a practical solution. The rehabilitation of ex-service personnel with persistent symptoms of neurosis is but part of the problem of coping with the great and rising incidence of neurosis in the community. We no longer speak of war neuroses, but rather of the neuroses of war, in an attempt to emphasize that the illness is essentially no different from that occurring in civilians who were not subjected to the emotional stresses of service conditions.

Neurosis is an illness with positive signs and symptoms. The diagnosis is not made by exclusion of physical causes. It is necessary to restate this fact to combat the attitude of contempt and indifference so often encountered by those whose aim is to provide adequate facilities for treating the neuroses in ex-service personnel and in civilians. The "pathology" or psychodynamics of the neuroses of war—the reasons why service personnel develop neuroses and why the symptoms persist during service and after discharge—has been exhaustively presented in the Beattie-Smith lectures of 1944 by Lieutenant-Colonel A. J. M. Sinclair.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on April 26, 1946.

It is here contended that the treatment of the neuroses should command the attention and intensive application of medical men to the same degree as any surgical or medical disease. No adequate practical attack on this problem has been made in Australia.

It is readily conceded that the ex-service man or woman who is ill deserves the best treatment medical knowledge and facilities can supply. For surgical and medical diseases the standard of treatment is admittedly good. For illness originating in persistent emotional disturbance, but still an illness no less real than a surgical or medical disease, treatment facilities are inadequate. The impression that the service man or woman who develops a neurosis is poor material anyway, that sympathy and even recognition only reinforce symptoms, and that a monetary encouragement, to retain symptoms in the form of a pension, must be avoided, is expressed in an attitude of over-compensation which has resulted in inactivity, an inactivity which cannot be glorified as masterly.

Everyone recognized the difficulties of physical and emotional adjustment encountered in the process of making civilians into efficient units in the services. It is obvious that any man or woman who has been in the services will find physical and emotional adjustment necessary on return to civilian life. Many of those released from the services were civilians in the full meaning of the word before enlistment. Many others have never occupied the place of grown-up men and women in the community. This process of becoming a civilian, either again or for the first time, is generally called rehabilitation.

In most discussions on the subject total emphasis is laid on occupation—a job for the service man, preference in employment. Not so much emphasis is placed on the type of work—work in keeping with the service man's aptitude and of his own choosing. A much quoted standard of successful rehabilitation is stated as follows: "The soldier's ability to return to and continue in a civil occupation without a pension." This standard is far from adequate as an indication of successful adjustment to civilian life, inasmuch as it ignores emotional adjustment in the social and domestic spheres.

Rehabilitation is not just a problem in economics. It is the subject and not the job that is to be rehabilitated. If we are to claim any advance on the methods employed after World War I, successful rehabilitation must comprehend a larger task than placement in a job. Attention must be given to the subject's interests and emotional life. The fundamental viewpoint accepts the subject as primary, his wants, interests, needs and capacities being the building stones with which to construct the edifice of rehabilitation. Many efforts fail because they are based on what is held to be good for the soldier rather than upon a careful evaluation of what the soldier wants and will accept.

Even in the field of placement in a job, the physically and emotionally handicapped ex-service man or woman is not given special consideration. There is nothing comparable with the scheme already implemented in Great Britain. The following are features of this scheme: (i) a disablement register for all disabled persons; (ii) a sheltered occupations register, positions to be filled only by persons on the disablement register; (iii) subsidized sheltered industries for the employment of personnel considered permanently subefficient.

Aubrey Lewis has written as follows: "It is easier to remember that illness can prevent work than that work can prevent illness—if only the work is well chosen." Work as a curative agent in the neuroses is accepted by all interested in the problem; but the work must be acceptable to the man in accordance with his aptitude and desires. Even if the man with persistent emotional disturbance is capable of full-time employment under existing competitive industrial conditions, and is fortunate enough to be placed in the ideal job, supervision and encouragement by an industrial welfare officer, social service worker or medical practitioner should be available.

Reconditioning centres have been advocated for those in whom emotional disturbance prevents full-time employment. Such centres would provide physical culture, occupational therapy, elementary trade training, gardening,

outdoor and indoor recreation, social service supervision and psychotherapy. The Royal Australian Air Force and the Australian Army Medical Corps successfully operated centres which correlated the techniques of the psychiatrist, the psychologist and vocational and recreational specialists.

For the ex-service man who is not making a satisfactory adjustment in the occupational, social or domestic spheres, the Australian Red Cross Society is making an earnest effort to provide adequate reconditioning facilities at the "Gibulla" rehabilitation farm, situated forty miles from Sydney. The resident manager and his wife are particularly suited to the supervision of the patients, and a social worker and psychiatrist visit the centre for a full day once a week. The accommodation, climate and environment are ideal and the recreational facilities varied and adequate. "Gibulla" is a good working model of the essential features of a reconditioning centre.

Annexes to factories with subsidized employment for the subefficient are a feature of the British scheme for rehabilitating the physically and emotionally handicapped. Work is treatment, and in any practical scheme for rehabilitating ex-service personnel with persistent neurotic symptoms, a close liaison must be maintained between the medical advisers and the organizations of employers and employees.

A question may be asked at this stage: Are the numbers of ex-service personnel who make a slow or bad adjustment in the occupational, social or domestic spheres worth worrying about?

Colonel A. Graham Butler, in "The Australian Army Medical Services in the War of 1914-1918", states that mental conditions overshadow all others among causes of disability during and after World War I. Neurosis was undoubtedly the greatest cause of long-term casualties in World War II. After his experience with troops evacuated from Norway and Dunkirk, T. A. Ross prophesied that this would be a "dyspeptic" war. The prophecy proved true. It was interesting to read the report of a recent conference of industrial welfare workers that the commonest cause of absenteeism in industry is dyspepsia. Many of you will have studied surveys on dyspeptic service men in Britain, United States of America and Australia. With amazing agreement these surveys reveal organic disease in less than 5% of personnel whose main complaint was of dyspepsia for a period of at least six months.

It can be stated with certainty that a large number of neurotic casualties have already returned to civil life with symptoms only superficially or partly remitted. In addition, large numbers have been discharged with a physical diagnosis, but the real basis of their illness is emotional. The psychosomatic nature of a proportion of medical, surgical and orthopædic casualties is seldom considered.

The incidence of "post-war" neurosis increases the scope of the problem. Many demobilized men and women think and feel that they have returned to an unfriendly environment. They are embittered, disillusioned, insecure and hostile. If the ex-service man finds that his service and sacrifice are "debunked", if he cannot preserve his feeling of personal value, "post-war" neurosis will be an even greater problem than the neuroses of war. Emotional dissatisfaction and threats to self-respect will force ex-service men to seek community of interest with other ex-service men. Any such segregation would be an indication of their failure to become civilians. Ex-service men's organizations have a valuable place in the community, but they should never be the only places where ex-service men can find understanding and a community of interest.

Follow-up surveys of men discharged from the services with a diagnosis of neurosis are appearing in the journals. Such a survey is in progress in Australia, but it is not yet completed. A small illustrative survey in South Australia has been published by Captain W. A. Dibden. The impression gained to date, however, corresponds with the conclusions of a survey in Britain: "The men had gone downhill as a group, were less usefully employed than before, less contented, less tolerable to live with, less healthy."

All psychiatric opinion is opposed to the granting of pensions to those whose illness is basically emotional in origin. It is heartening to note that lay organizations are now accepting this sound principle. On the other hand, to quote Captain Dibden, "neglect will not cure".

Discharged service men are naturally consulting their private doctors. As these practitioners must rely entirely on the patient's story, and have no access to service medical records, the advice in many cases is most damaging. Dyspeptics are having tests repeated and diets and medicines prescribed; "effort syndrome" patients are being rested in bed for as long as three months; sufferers from post-traumatic headache are being protected and restricted in their activities. The general practitioner with a psychosomatic outlook could give immense assistance in coping with the problem of neurotic ex-service personnel.

Owing to the provisions of the act under which it works, the Repatriation Commission grants entitlement to treatment to a small percentage only of neurotics. It is being advocated that acceptance for service should be an entitlement to treatment. The opinion, only partly true, that most service neurotics would have reacted to maladjustment in the civilian sphere sooner or later should not debar from adequate treatment men and women whose illness was precipitated by emotional stress during service. All neurotics, ex-service and civilian, are entitled to adequate treatment.

Apart from the out-patient facilities provided by the Repatriation Commission, ex-service neurotics have to rely on the already overcrowded out-patient departments at civilian hospitals. In this State limited in-patient accommodation is available at the Royal Prince Alfred Hospital and at Broughton Hall. Private hospital accommodation for the emotionally ill is almost non-existent.

Neurosis in the community is a widespread and disabling illness. Expansion of present facilities for prevention and treatment is an urgent necessity. The ideal general hospital would provide adequate out-patient and in-patient accommodation for patients with neurotic and psychosomatic disorders. It will be many years before this ideal can be achieved in Australia—the psychiatrists will be the last to obtain beds in general hospitals. To quote from a report by Lieutenant-Colonel A. J. M. Sinclair:

> The lack of appreciation of the necessity of facilities for such treatment is due in part to the small number of physicians properly trained in psychological medicine, to inadequate education in psychological branches of medicine among medical students, and also to a state of profound unenlightenment amongst many responsible public bodies. The principles of treatment of the psychologically disordered differ in some respects from the treatment of physical ailments insofar as the treatment includes not only medical care and supervision, but proper social and industrial re-alignment in those affected. Any consideration of plans for treatment must therefore include plans for improved medical, social, psychological and industrial co-operation.

The only practical approach seems to lie in the establishment of a separate psychological clinic in each State. Created primarily and promptly for the treatment of the large numbers of ex-service personnel with persistent symptoms of neurosis, these clinics could be expanded to deal with psychological disorder in the community. The main object of this paper is to present this plan for your consideration.

An investigation in England revealed that less than 25% of patients from a large general hospital were cured. There can be no doubt that unrecognized and untreated emotional disturbance is largely responsible for the poor result. "Dyspepsia" alone is a challenge to the medical profession. The psychosomatic approach must prevail in all branches of medical practice.

In summary, large numbers of ex-service personnel are failing to make a satisfactory adjustment in the occupational, social and domestic spheres. Some of these were discharged from the services with symptoms of neurosis only superficially or partially relieved. Others were discharged with a physical diagnosis, but their real disability

is emotional. Many others, including recovered personnel, did not develop overt neurosis while on service, but are breaking down because of lack of adjustment to psychological, social and industrial hazards.

Existing facilities for treatment are inadequate. Apart from a humane interest in the subject, the welfare of the community must be served by salvaging ex-service personnel with persistent neurotic symptoms and reduced efficiency. Neurosis in the services is regarded as an "infectious disease". It is equally infectious in the civilian sphere, inasmuch as the neurotic adversely affects those among whom he lives and moves.

Treatment facilities must be provided promptly. The problem of ex-service personnel, partially or totally disabled by persistent emotional disorder, is urgent and extensive. Only a small percentage are entitled to treatment from the Repatriation Commission. The out-patient departments of civilian hospitals cannot cope with them. Separate clinics for counselling and psychotherapy appear to be the only practical solution.

Let me conclude with a question: As medical men, do we acknowledge any obligation to the demobilized service man and woman over and above the duty of the individual citizen?

THE REHABILITATION OF WAR NEUROTICS.[1]

By H. HASTINGS WILLIS,
Senior Medical Officer, Department of Repatriation, Sydney.

WHEN I was asked by the Section of Medicine to follow a psychiatrist this evening with some remarks on the rehabilitation of war neurotics from a general practitioner's point of view, I had some diffidence in accepting the invitation. Firstly, I feared that by provocative remarks I would upset the customary serenity of a British Medical Association meeting and, in the second place, my close association with such work, over more than twenty years, unfits me to speak dogmatically with assurance on the subject.

For the onlooker sees most of a game. I have been deeply involved in this game—a sort of football scrum in which I have been bumped and knocked about, sometimes accidentally, sometimes intentionally, and bespattered with mud. This colours my outlook on the problem.

When I spoke in this hall six months ago to a gathering of psychiatrists, I was accused, in the discussion which followed, of being pessimistic and a defeatist. I trust I am not that, but I must admit that experience has dampened my early enthusiasm. I hope you will discount any pessimism in my remarks accordingly.

A further difficulty, and my most serious one, arises from the fact that I am a public servant whose duty, like that of my fellows, is to implement, not formulate, policies, and this is as much a political and social problem as a medical problem that we are to discuss. However, it has its medical aspects, which is the justification for its discussion at a medical meeting like this, and these medical aspects concern psychiatry.

Some Definitions.

Now in discussing any psychiatric problem it is as well to start with a few definitions. Psychiatrists speak a language of their own. Unfortunately, the various warring sects of psychiatrists speak different dialects and, worse still, a word may have a different shade of meaning in one dialect from what it has in another.

From the point of view of rehabilitation let us consider three terms in common use: temperament, personality and character.

Temperament.—By temperament I mean the inherited factor in mental disorder. The general practitioner and his patients have no doubt about the existence of an inherited factor in mental disorder. The psychiatrist is not so sure about it. Some will deny its existence and ascribe to parental environment what I would ascribe to heredity. Cases will be cited of adopted children who show the temperamental characteristics of their foster-parents, not of their natural parents, but, contrariwise, it is possible to show some who manifest the characters of their natural parents. It is all a matter of the relative predominance of temperament and environment. These two, in varying proportions, are responsible for personality.

Personality.—The inherited temperament of the child is modified by the stresses and strains of its early environment in such a way as to result in a personality which it now seems generally agreed is fixed before school age. As the child cannot choose its parents or its early environment, it cannot fairly be held responsible for its personality. Our legal friends have fixed the age of legal responsibility at six. Moreover, at that age a child begins his education. The effect of education upon personality results in his character.

Character.—I believe it an error of the age to minimize the importance of character in cases of abnormal behaviour. Reading through case sheets of war neurotics, I have been struck by a number of men sent to hospital for treatment of defective personality who in the first German war would have been sent to a court martial. The practice has apparently been to give the defaulter the benefit of the doubt and to ascribe his misdemeanour to defects of personality rather than to faults in character. This is in line with common usage in civil life today. Delinquency, previously regarded as due to bad character and meriting punishment, is now considered a disorder of personality to be treated in a hospital. This may be just to the individual, in some cases, but is it in the interests of the general public and the nation?

In the formation of character many educational factors play their part. The child learns at school as much from his fellows as from his teachers, and education in its widest sense goes on throughout life. The individual can and does determine to a large extent his education and, therefore, is rightly considered responsible for his character. But the earliest and more potent influence must necessarily come from his teachers, and here I would stress the importance of strong character in the teachers.

In the old days parents chose a school largely because of its masters. Schools were known as Greenway's School, Hutchin's School, Herriot's School *et cetera*, which indicated that in that age the controlling headmaster was considered the most important part of the school and parents chose for their children the school whose teachers were of strong character. For most persons today, the teacher is chosen by the State and, as far as I am aware, character is not one of the things which is taken into consideration in the selection of candidates for entry to our State teaching service. Scholastic attainments, I understand—and I speak subject to correction—are the sole criteria of suitability. Once selected for training, a student is taught how to impart knowledge rather than to mould character, and anything in the way of indoctrination it is fashionable to condemn.

I fear I am here treading on dangerous ground, for education in Australia is partly a political issue. Suffice it for me to point out that many who by strength of character served their generation well in the history of the British race, were enabled to do so because of early indoctrination by a teacher of strong character. The boy, we were told, is father to the man, and this is still essentially true, though character may continue to be moulded, even until old age.

In the rehabilitation of war neurotics my experience has been that character counts for much more than personality, and I feel that psychiatrists would get better results in their work if they paid more attention to character and less to personality which, if the doctrine underlying these definitions is true, can be but little altered, having been

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on April 26, 1946.

fixed before the age of six. A person of strong character, no matter how poor his personality, can be expected to rehabilitate himself with a minimum of assistance, but a person of poor character is a difficult person to help.

War Neurosis.

I take it that the word neurosis is not to be used tonight in any technical sense as distinct, for instance, from a psychoneurosis or a psychosis.

The general public and the general practitioner understand by it "uncertified mental disorder", and I do not think it helpful to pander to the susceptibilities of our patients by assuring them that they are not "mental", only "neurotic". Neurosis is a mental disorder and we should be frank about it.

Medically it is generally agreed that the war neurotic does not differ essentially from the civilian neurotic. In few cases does neurosis arise directly as a result of battle stress or conditions peculiar to warfare. Most neuroses arising in the services could equally well have arisen in the same way in civil life. But politically there is a world of difference between the war neurotic and the civilian. By all means let us treat them with consideration and kindness, but firmness in dealing with war neurotics is essential. For political reasons this firmness is very difficult to maintain. The very thorough way in which neurosis was dealt with in the services during this war led some optimists to believe that there would be no neurotics to be dealt with after discharge. Things were not going to be as they were after the 1914-1918 war; things were going to be done better this time; but experience shows that the same old mistakes have been made, the same old failures of readjustment have recurred and we have with us as big or bigger problems than we had in 1920. Some of these neuroses will have first occurred on service and persisted despite treatment. Others will have been successfully treated during service, but will have recurred at varying intervals after discharge from post-war causes. Here I would mention the doctrine of facilitation. This doctrine, which was propounded by soldiers' friends after the 1914-1918 war and supported by some eminent members of our profession, is that one nervous breakdown makes a patient more likely throughout later life to suffer another breakdown from other causes. Hence it was contended that a case of neurosis occurring many years after the war from such obviously non-war causes as domestic worries or financial hardships could be regarded as due to war service if the sufferer had been treated for a neurosis on service. I do not know whether psychiatrists in general will accept this doctrine. We know that facilitation is sound doctrine in physiology. Is it sound in psychology?

Another type of case which we will meet some years after the war is that of the neurotic to whom the propagandists apply the term "delayed shell shock". Of course, it has nothing whatever to do with shells and is not shock. As we saw it in the depression of the thirties it was an ordinary sort of civilian neurosis occurring from very ordinary civilian causes, and we can fairly say that it had no relation to previous war service. But when a neurosis first occurs reasonably soon after discharge from the services, a broad view might regard the conditions of service as a factor. Many men well adjusted in civil life were unable to adjust to army conditions. Likewise, many ill-adjusted before enlistment took kindly to life in the services, but find themselves unable to readjust themselves to civil life after discharge. This type is quite common and its attributability to conditions of service is a matter which can fairly be argued.

Now a word as to "doctor-made" neurotics. As a profession we must take the blame for many cases of neurosis. Bad diagnosis or an uncertain diagnosis will often be the groundwork upon which a neurosis is built. Particularly is this the case with patients complaining of cardiac symptoms. Many a man has been made miserable for life by being wrongly diagnosed as suffering from heart disease.

So impressed have I been over the years by the harm done in this way that I say without hesitation that no diagnosis of organic disease should be made without very good cause. If there is any doubt, give the patient the benefit of the doubt and regard the condition as neurotic. I have never known of any harm coming to a sufferer from organic disease who was treated as neurotic, but I have seen a lot of miserable patients whose unhappy lot has been due to a neurosis having been mistaken for organic disease. And, of course, neurosis and organic diseases can and often do exist together, sometimes in association, sometimes as two distinct disorders, for a neurotic can suffer from organic disease as easily as a person of normal mentality.

The first World War was a cardiac war because doctors were "heart-minded". This last war has been called a dyspeptic war. The present generation of medical graduates seems to be stomach-minded. We should not blame them; we behaved similarly in our day. When, as recent graduates, we joined the services, we were sent, being young and active, to field units. We had learned for our final examinations all about the up-to-date discoveries of Mackenzie and Lewis and the insignificance of murmurs and the differentiation of cardiac irregularities. We viewed with scorn our seniors, who spoke of "those irregular hearts". We knew them as cases of auricular fibrillation, which was serious, or sinus arrhythmia, which was a "certain sign of a healthy heart", and so on. When, therefore, in a forward area the soldier presented himself on sick parade, suffering from tremor, sweating, palpitation, loss of appetite and a "churned-up feeling inside him", being cardiac-minded, we seized upon tachycardia and then by a few suggestive questions "elicited" a cardiac syndrome which we labelled "D.A.H." Likewise, in this war, the present generation, being stomach-minded, has seized upon the lack of appetite and the churned-up feeling, and by a similar process of suggestive questionings has obtained justification for a diagnosis of dyspepsia. Once diagnosed as suffering from dyspepsia, the patient went off to hospital, where specialists, more numerous in this than in previous wars, subjected him to elaborate investigation and sent him from radiologist to radiologist in the hope of finding one obliging enough to diagnose duodenal ulcer. If none obliged, the patient was labelled "functional dyspepsia" and handed over to the psychiatrist for treatment. After discharge these patients with duodenal ulcer and functional dyspepsia have come to crowd our out-patient clinics in just the same way as those with effort syndrome and other cardiac neuroses did after the first World War.

This leads us to consideration of the fixation of neuroses. General practitioners are frequently blamed by specialists for fixing neuroses. The psychiatrist likes to keep neurotic symptoms wandering. Apparently the idea is that if the symptoms shift from organ to organ they can be more easily chased away. This is probably correct, but I see some advantages in fixing neurotic symptoms on systems where they can do least harm. In this respect let us consider for a moment functional dyspepsia. I do not know whether the psychosomatic theory of the causation of duodenal ulcer has as yet received any considerable support in the profession, but I have seen some cases which suggest that a functional dyspepsia can become a case of duodenal ulcer. And these are cases in which the diagnosis has been changed, not because of radiographic shadows, but because of such unequivocal occurrences as hæmorrhage or perforation. If, therefore, a neurosis by being fixed on the stomach can result in a serious surgical catastrophe, then there are advantages in fixing a neurosis somewhere else than on the stomach.

Rehabilitation.

One hears the term rehabilitation frequently these days and its exact meaning is not clear. Purists prefer the word "reablement", and when applied to orthopædic patients this word is, I think, preferable. The neurotic, however, requires something more than reablement—restoration of working capacity. He requires to be dressed up again as a civilian. The army took him from civil life and made a soldier of him; our task is to make a civilian of him again.

The idea which underlies the term is the modern conception of a doctor's duty to his patient. It is no longer sufficient to save life and ease pain, nor even to diagnose disease correctly and to treat it. We must go further and remake our patients as happy, useful members of the community. From the patient's point of view rehabilitation is an active not a passive process. Many speak as if rehabilitation could be "pumped" into an individual while he rests at ease. Nothing is further from the truth. The neurotic must rehabilitate himself; our task is to help him.

Who are to be the rehabilitators? The psychiatrist wants to do this work, and by all means let him do it; but let him realize that he is only the leader of a band of workers, each of whom is as important as himself.

Rehabilitation and treatment should go hand in hand, not one follow the other. This cardinal error of the 1914-1918 war has been repeated in this last war. Patients have been treated in the artificial environment of elaborately equipped hospitals and on discharge have found themselves unable to cope with actual conditions of life.

For success psychiatrists must be "socially minded" and also temperamentally fitted for this work. In my experience those psychiatrists have done most good for their patients who have taken a sympathetic social interest in them as human beings, not only as interesting cases.

The second member of the team is the employment officer. His task is to find a patient work and to keep him at it. I am aware that finding employment is not the whole part of rehabilitation, but it is 90% of it. Regular congenial, remunerative employment is the main requirement of the war neurotic. To find the job is relatively easy, but to keep the man in the job is more troublesome and requires our employment officer to be something of a welfare officer as well.

The third member is the social worker. If the coal miners had allowed this discussion to take place on due date some five months ago, I should have roundly condemned social workers, as my experience of them towards the latter part of last year was rather unfortunate. I have had time to relent and on contemplation remember that for many years my colleagues and I have found it useful to obtain reports from persons we call investigators on the home conditions of some of our patients, and I am aware that for many years social workers have been employed with advantage in the mental hospitals of this State. The truth is that a good social worker is very useful, but a bad one is worthless or harmful. What, then, constitutes a good social worker? It is usual to stress the necessity for training, but I suggest that temperamental fitness for the work is of equal, or greater, importance.

The lay psychologist is usually given a place in the team. In the services he has been used as a psychometrist, and I am told was useful. He has, however, labelled as mentally deficient many indignant patients who have seemed to me quite ordinary persons. We must remember that a mental age of sixteen is equal to full adult intelligence and that a mentality corresponding to age twelve is quite enough for most occupations in life. The standard of normality seems to have been set too high.

In this city lay psychologists are increasing in number and are prepared to treat mental disorders. It would seem that psychiatrists have been carried away by modern physical methods of treatment, such as insulin therapy and electric convulsive therapy, and have tended to neglect old-fashioned psychotherapy. This vacuum the psychologist is rushing in to fill and is receiving a good deal of recognition in some quarters. For instance, I was recently asked by a well-informed layman: "Does the Repatriation Department treat its patients psychologically or psychiatrically?" If the neurotic is not given the psychotherapy he needs by the psychiatrist, he will seek it from the psychologist.

So much for the medical rehabilitation team. Outside the team there are rehabilitators who can do their part and effect more than can the team itself.

The employers and the foremen in industry need to be sympathetic toward the neurotic. I can recall many instances in which the rehabilitation of the neurotic was largely, if not solely, due to a little consideration from his superiors. Fellow workers need also to be tolerant. The worker who says that Bill is mad and should see a doctor or that he does not know why "Bill is not put off on compo., as he is only a nuisance round the place" does not help, but the worker who says to Bill, "Why don't you pull yourself together and play the game" discourages neurosis while tolerating it. A sympathetic employer and tolerant fellow workers will often do more for the neurotic ex-service man than will any medical team or government organization.

Where does the family doctor come in in all of this? At the beginning and at the end certainly, for he it is who should choose the case for the team, and it is he who must follow on the work of the team; in many cases he can be the team himself. Many neurotics do not require more treatment than simple explanation, encouragement and a little suggestion. This the family doctor is well able to give. As a social worker the doctor has unique opportunities of ascertaining the heredity, the social background and the environment of his patient.

I have no scheme of rehabilitation to put before you. This is the season for such schemes and no doubt we shall hear of many of them in the near future. I agree with the previous speaker when he says that the guiding principle of any scheme must be the need of the patient. It is a pleasant fantasy to sit in an armchair before a winter fire and conjure up grandiose schemes for the rehabilitation of neurotics, but it is a futile pastime. Let us meet the patients' need, and as patients vary, their needs will vary. If in doing so we evolve a scheme, no harm is done, for schemes are useful administratively; but any scheme to be practical must be a loose and ill-defined scheme capable of modification as required. Such schemes will not be needed to deal with all our neurotics; most of them, fortunately, will rehabilitate themselves. These are the ones of adequate character. At the other extreme there are some of poor characters, who will not cooperate and for whom little can be done. These are the ones responsible for the views of many experienced laymen that rehabilitation for neurotics is a waste of energy and public money. This certainly is true of some, but they are in the minority. Between the two extremes are those, about 20%, for whom something should be done. It is fashionable at present to urge that these be placed in institutions or farm homes. For some cases these institutions are desirable, perhaps necessary, as treatment sometimes cannot be given elsewhere.

After the 1914-1918 war rehabilitation homes were provided. Some of us who saw their activities approved of them; some were not impressed by their results. Another generation is repeating the experiment after this last war, and we shall be interested to see their results.

For myself, I prefer the out-patient clinic as the place for the treatment and management of these patients. There the patient can be treated while on his job. This is better than to attempt rehabilitation in the artificial atmosphere of a hospital or a pleasant country rest home.

Obstacles to Successful Rehabilitation.

1. Besides positive action much can be done in a negative way to help the neurotic by removing obstacles to his successful rehabilitation. The ex-service man must rehabilitate himself. Let us make it as easy as possible by removing the obstacles in his path. First and foremost of these is the well-meant but misdirected propaganda of his publicist friends.

Action and reaction are equal and oppositive, we learned at school, and any successful propaganda which secures benefits for any particular class of service man, neurotic or other, provokes a reaction of hostility, often hidden, but none the less harmful, against them. Even the preference of employment law, so generally approved, has proved a disadvantage to many able men, who have found the consequent hostility of their fellows a handicap to their advancement in life, which outweighed the benefits they derived from that legislation.

2. The attitude of the public towards neurotics is wrong. There is at present a tendency to cultivate neurosis and to exaggerate the problem of the neurotic. I heard of a captain of industry who recently said that neurosis was the greatest problem in industry today. I find it hard to believe that. Troublesome as a neurotic is, both at work and at home, nothing is to be gained by exaggeration; often a healthy disregard is the better attitude to adopt.

3. The attitude of the Press is often not helpful. Journalists seem to have an affinity with psychiatrists and their patients. We have noticed how our psychiatrists fill our medical journals with many pages of prose expressing noble sentiments in strange words and phrases. Their patients—at least when in trouble—likewise fly to the journalists. These ensure for the disgruntled an excess of publicity which satisfies their vanity, but gives them very little real help.

Pensions and Compensation.

I get a little personal satisfaction from the general acceptance these days by our profession that pensions or compensation or any other benefit are serious obstacles to the recovery of the neurotic. It was not always so. When my colleagues first pointed this out some fifteen years ago they received a good deal of abuse from soldiers' friends and uncomplimentary terms were applied to them. Since then the profession generally has had ample opportunity in dealing with claims under the workers' compensation acts of appreciating the harmful effects of money payments of all sorts in these cases. Unfortunately, the general public and our legislators are not as well aware of these harmful effects as our medical men, so the law still says that neurotics must be compensated, just as the sufferers from organic disease, if assessable incapacity is present. Thus we see the demands of the present champions of the ex-service men, that the neurotics be treated in the same way as the sufferers from organic disease, have in this respect at least been met many years ago.

The Stigma of Mental Disorder.

Let me close my remarks by asking you to consider for one moment the stigma of mental disorder, perhaps the greatest of all obstacles to the successful rehabilitation of the neurotic. It is futile to deny its existence, as some would. It is very real and has as its foundation the general public's recognition of the hereditary element in mental disorder.

It is quite easy to find many instances of patients who have refused to undergo shock therapy, for which their symptoms clamoured aloud, because of the stigma which they thought would thereby be placed upon themselves and their families.

Recently I became aware of a case in which a man's matrimonial prospects were shattered when the fiancée's relatives learned that he had been treated with shock therapy in the army. What, then, can we do to remove the stigma? The current practice is to run away from it. We see this in the demand that war neurotics should not be treated in mental institutions, but in some special institutions or in special wards of general hospitals, to which it is assumed no stigma is attached. But the patient carries the stigma with him; it is on the institution merely because of the patients in it, and wherever the mental patient is housed in any special institution or part of an institution, the stigma will come to rest. I have found it is possible to avoid it by admitting patients into ordinary wards of ordinary general hospitals, but this is not always convenient from an administrative point of view, and is considered undesirable by many psychiatrists. I suggest that those who are now trying to diffuse the stigma as widely as possible are approaching the subject from a better angle. Let us not draw fine distinctions between neurotics and psychotics; let them all be cases of mental disorder. Let the general public realize that mental disorder in some form or other is part of the make-up of each one of us. In short, let us all bear the stigma, then it will cease to be a disability to anyone.

THE DETECTION OF THE CYSTS OF ENTAMŒBA HISTOLYTICA IN THE FÆCES BY MICROSCOPIC EXAMINATION.

By A. T. H. MARSDEN, M.D.,
Melbourne.

IN the acute stage of amœbic dysentery it is relatively easy to detect the vegetative forms of *Entamœba histolytica* in the fæces, and it is reasonable to suppose that few such cases will be missed. But when the acute stage subsides, either naturally or as the result of treatment, and conditions have become unfavourable for the further growth and multiplication of the amœbæ, those amœbæ which are deep in the tissues become surrounded by fibrous tissue and remain there inert, as may be seen in sections of amœbic granulomata, while those which are in open ulcers change into precystic forms and then into cysts which are discharged in the fæces.

To detect these cyst-passers is a different matter, because the cysts are often passed only at intervals, and even when they are present in the stool they may be present only in small numbers, and no blood and mucus are present to indicate which part of the stool to examine. Further, although the stools of patients with such chronic amœbic infections will frequently not be normal in appearance, this is not necessarily so, and in any case there is nothing really pathognomonic about them. If cysts are not seen on microscopic examination, there is no indication whether or not further stools should be examined, for erythrocytes, pus cells, epithelial cells or Charcot-Leyden crystals are not associated with the presence of *Entamœba histolytica* cysts, nor have I been able to find anything which is so associated.

It has been suggested in an American paper that the cysts of *Entamœba coli* serve as an index of *Entamœba histolytica* infection, and certainly it is my own impression that *Entamœba histolytica* cysts are more frequently associated with *Entamœba coli* cysts than with anything else; but I have never been able to find any positive correlation between the presence of *Entamœba coli* cysts and *Entamœba histolytica* cysts. For all these reasons cyst-passers may easily escape detection.

The problem of their detection was brought to my notice when I was in charge of the clinical pathology laboratory at the Nakompaton prisoner-of-war hospital, Thailand. We had in the hospital many men with amœbic dysentery under treatment, and the question arose, how many stools should be examined as one of the criteria of cure. Different authorities recommend different numbers of stool examinations for this purpose, and agree only in appearing to have no rational basis for their choice. What proportion of cyst-passers will be detected in any given number of stool examinations appears to be unknown, though there seems to be a tacit suggestion that all will be found by a few examinations, which is certainly untrue.

The same problem recurred in a slightly different form when we were asked to examine stools from men not suffering from dysentery, and without any history of dysentery, in an endeavour to ascertain the true extent of amœbic infection. The routine examination of stools of cooks raised the same question again. In both cases a certain number of cyst-passers were detected; but how many were missed, and what was the true number of chronic amœbic infections?

This problem appeared to require investigation and an attempt to evaluate the significance of a negative result. In an endeavour to do this it was decided arbitrarily to examine six consecutive stools from each patient and when sufficient data had been obtained, to submit our results to statistical analysis in the hope that some light might be shed upon the problem.

The analysis was made by Mr. H. Fairfield Smith, statistician to the Rubber Research Institute of Malaya, whom I was fortunate enough to have on my staff at the time. All the opinions and conclusions herein expressed

are based on that analysis, which is printed as an appendix to this paper. As a result of this work the position has been somewhat clarified; but a full answer has not been found, and in my opinion cannot be found.

In all attempts to demonstrate an infection, the results may be considered from either of two different points of view: that of clinical medicine concerned with the individual patient or that of preventive medicine concerned with the population as a whole.

Let us take first the point of view of the clinician and consider the individual patient. Here the problem is simply this: is this patient infected or not? It seems a reasonable question; but unfortunately it cannot always be answered with certainty. The way in which the question is frequently put by both clinician and patient is: "How many negative results to stool examinations are required before we can be satisfied that there is no infection with *Entamœba histolytica*?" The answer to this is an infinitely large number, or at least so large a number as to be quite impracticable. Perhaps it would be more accurate to reply that the answer depends on the degree of infection and on the number of cysts and the frequency with which they are passed. A positive result has one simple and definite meaning; but a negative result may mean either that the patient is not infected or that the patient has a degree of infection insufficient to be detected in the number of stools examined. In this connexion it may be worth while emphasizing that in a condition in which cysts are so often passed intermittently, a considerable degree of infection may fail to be detected at a single examination. A few concrete examples may illustrate the position.

One cyst is almost certain to be seen in two stool examinations from a patient passing 1,000,000 cysts per day, while there is an even chance of detecting a cyst in two stool examinations from a patient passing 100,000 cysts per day; but to obtain an even chance of detecting even one cyst from a patient passing 1,000 cysts per day, no less than 220 stools must be examined.

The important point to realize is the limitation of stool examinations for the detection of *Entamœba histolytica* cysts and, having decided on a certain number of stool examinations, to appreciate the significance of a negative result. The number of stools to be examined should be chosen so as to give a maximum number of detections for the work involved. If an attempt is made to detect the lesser degrees of infection, the number of stools to be examined increases out of all proportion to the extra number of cyst-passers detected.

How, then, are the lesser degrees of infection to be demonstrated? Use of a concentration method for cysts or animal inoculation will help if cysts are actually present in the stool; but such patients do not excrete cysts every day, and many of the stools will not contain any cysts at all. More, therefore, is to be hoped from an intradermal reaction or a complement fixation test.

When the problem of the detection of chronic amœbic infection is considered from the epidemiological point of view, it becomes a little easier. The question now is not is this individual patient infected, but what proportion of the population is infected. If, for example, there is an even chance of detecting a certain degree of infection by a certain number of examinations, that may not give any useful information about an individual patient, but when applied to a population this "even chance" has the meaning that about half the subjects with that degree of infection will be detected. Further, if as a result of a series of such examinations a certain number of cyst-passers are detected, they will be the more heavily infected, and those who are missed, being the more lightly infected, will, other things being equal, be less dangerous.

What degrees of infection and what proportion of those degrees it is desired to detect can be decided beforehand and the number of examinations chosen accordingly. The question of what proportion of the total infected population will be detected by any given number of examinations cannot, in my opinion, be answered; the proportion must vary from time to time and place to place, as it depends on the proportions of the varying degrees of infection. At Nakompaton we examined six consecutive stools from 1,015 patients, who it was hoped had been cured of amœbic dysentery, and about 30% were found to be still infected. In this case, therefore, six consecutive examinations cannot have detected less than one-third of the true number still infected, and as it is not reasonable to suppose that all patients were in fact still infected (all the patients were clinically recovered), the proportion of infected patients detected must be higher than this; in my opinion at least half were detected. It is worth noting that two consecutive examinations would have demonstrated only about half of those found at six examinations.

From a consideration of the analysis of our results it is possible now to recommend an optimum number of stool examinations, selected for definite reasons, for the detection of chronic *Entamœba histolytica* infections. The best number is, in my opinion, four stool examinations at intervals of one week. If for any reason this is impracticable and consecutive examinations must be made, then six examinations should be made. Such a series will almost certainly detect any patient passing 1,000,000 cysts a day or more and give a three to one chance of detecting anyone passing 100,000 cysts per day, while, of course, there is a rapidly decreasing possibility of detecting lesser degrees of infection. From the epidemiological point of view practically all carriers passing 1,000,000 cysts a day or more will be detected, about 74% of those passing 100,000 cysts a day, about 12% of those passing 10,000 cysts a day, and possibly (according to the number examined) a few of the more slightly infected. The above figures, of course, are true only if a sufficiently large number of people are examined.

Conclusions.

1. Diagnosis of a chronic infection with *Entamœba histolytica* by microscopic examination of the fæces for the presence of cysts depends for its success or failure on the number of cysts passed and on the frequency with which they are excreted.

2. There is no way of detecting all chronic amœbic infections by stool examinations, and detection of any but a small minority of patients passing 10,000 cysts or less per day is impracticable.

3. Stool examinations should not be made on consecutive days, because most cyst-passers excrete their cysts for only a few days at a time and at random intervals. From this it follows that stool examinations on two or three consecutive days only do not give a true picture of the position.

4. Four stool examinations at intervals of one week are recommended as being the most efficient.

5. Examination of more than one preparation from the same stool increases the chance of detection so little as not to be worth while. I do not refer to cases in which atypical cysts are found and further preparations are examined in the hope of finding typical cysts.

6. It is emphasized that all figures given are lower limits, and better results than those quoted in the text should usually be obtained.

Acknowledgements.

I wish to take this opportunity of recording my thanks to Lieutenant-Colonel A. E. Coates, Australian Army Medical Corps, Australian Imperial Force, officer commanding Nakompaton prisoner-of-war hospital, for his constant help and encouragement to the pathology department of that hospital.

Appendix: Frequency of Detection of Cysts of Entamœba Histolytica in Stools from Patients Apparently Recovered from Amœbic Dysentery.

By H. FAIRFIELD SMITH.

I: Data.

The observations to be described were obtained in the pathology laboratory attached to the prisoner-of-war hospital camp at Nakompaton, Thailand, during the months

from November, 1944, to July, 1945. In the major part (Section II (*a*) to II (*d*)—Statistical Summary) of the observations here reported, the patients were men who had suffered from amœbic dysentery sometime during the preceding two years, but had become sufficiently healthy so that it seemed possible that they might be now free of the disease. The plan was that six stools passed on successive days should be examined for each patient. For some men, however, fewer than six stools were submitted. The numbers included in this survey are shown in Table I.

Section II (*b*) includes the same series as Section II (*a*), in addition to 253 patients for whom observations were stopped when one "positive" specimen had been found. In the statistical summary which follows, adjustments have been made for the numbers which failed to be six times observed. Such adjustments are trivial, and so will not be described in detail.

Technique.—About one cubic millimetre of fæces from various parts of the stool, but principally from the surface if the stool was formed, or of blood and/or mucus if these were visible, was taken up on a bamboo stick, mixed with a drop of normal saline solution on a microscope slide and covered with a cover slip, usually about eighteen millimetres square. The whole was searched with a 2/3 inch objective and selected areas were examined with the 1/6 inch objective. Slides were recorded as showing positive results only when there could be no reasonable doubt about the species of amœba observed. The criteria principally used, in order of emphasis, were as follows: size and shape of chromidial bar, thickness of wall, size and visibility of nuclei. Occasionally iodine staining was used for observation of nuclei; but we had no facilities for fixing and staining with hæmotoxylin *et cetera* as would have have been desirable to check doubtful findings.

Comment.—Unfortunately the records are not so accurate as we would wish, because there are grounds for suspecting that some men—some who hoped to be passed as fit for work, as well as some who wanted still to appear ill—submitted stools other than their own. We believe, however, that the great majority of the specimens have been honestly delivered, and the general trend of the data, if not its details, may not be appreciably affected.

II: Statistical Summary.

(*a*) Frequencies of positive findings in the series of six stools submitted by each patient were as shown in Table II.

It was to be expected that the "6" group would be greater than the "5", because in that group occurred all those patients with so heavy an infection that cysts were observed every day, however long the series might be continued.

(*b*) The initial observation of cysts in successive specimens was as follows (Table III).

The somewhat surprising feature of these data is the equality of frequencies appearing at the fourth, fifth and sixth stools, apparently suggesting indefinite continuation of positive findings if the series was to be extended. That this is a constant feature of our data is shown by the fact that the relative figures remain approximately the same when evaluated for subsamples covering only three months.

(*c*) The observations on successive days were associated as shown in Tables IV and V.

Table IV shows the frequency with which all positive results occurred on successive days, as compared with other arrangements throughout the six stools examined. The "calculated figures" show the distribution between these two classes to be expected if the observations were distributed at random throughout the six stools.

TABLE IV.

Positive Findings.		All Results Positive on Successive Days.	Others.	Total.
Two positive results in six stools.	Observed.	9·0	14·0	23
	Calculated.	7·7	15·3	
Three positive results in six stools.	Observed.	10·0	16·0	26
	Calculated.	5·2	20·8	
Four positive results in six stools.	Observed.	7·0	11·0	18
	Calculated.	3·6	14·4	
Two positive results in five stools.	Observed.	8·0	2·0	10
	Calculated.	4·0	6·0	
Three positive results in five stools.	Observed.	5·0	4·0	9
	Calculated.	2·7	6·3	

(5d.f.) = 18·35.

There is therefore a significant tendency for persons with moderate or light infections to pass over periods of a few days larger numbers of cysts than their average.

Analogous classification of the same data with respect to association of negative findings is as shown in Table V.

This appears to indicate that the periodicity of quiescent periods is at random.

TABLE I.

Number of Patients.	Number of Stools Observed per Man.						Total.
	6	5	4	3	2	1	
Section II (*a*)	537	135	50	19	13	8	762
Section II (*b*)	665	164	60	35	37	54	1,015

TABLE II.

Positive Findings.	Frequency of Positive Findings out of Six Stools.						Total.		Grand Total.
	1	2	3	4	5	6	Positive.	Negative.	
Number	77	44	38	33	14	27	233	529	762
Percentage	33·0	18·9	16·3	14·2	6·0	11·6	100		

TABLE III.

Positive Findings.	Serial Order of Stool in which Cysts were First Observed.						Total.		Grand Total.
	1	2	3	4	5	6	Positive.	Negative.	
Number	112	65	44	27	30	29	307	708	1,015
Percentage	36·5	21·1	14·5	8·8	9·6	9·5	100		

(*d*) The associations of cysts with ancillary characters—namely, epithelial cells, pus cells, red blood corpuscles and Charcot-Leyden crystals—were as shown in Table VI.

Some negative association appears to be indicated. Deviation from random expectation is in the same direction when the data are classified with respect to only single ancillary characters, or subdivided with respect to intensity of infection (as indicated by the number of days on which positive findings were obtained). I offer no explanation.

Among patients passing both cysts and ancillary characters there is no observable tendency for both to appear either on the same or on different days.

TABLE V.

Negative Findings.		All Negative Results on Successive Days.	Others.	Total.
Two negative findings in six stools.	Observed.	7·0	11·0	18
	Calculated.	6·0	12·0	
Three negative results in six stools.	Observed.	6·0	20·0	26
	Calculated.	5·2	20·8	
Four negative results in six stools.	Observed.	5·0	18·0	23
	Calculated.	4·6	18·4	
Two negative results in five stools.	Observed.	4·0	5·0	9
	Calculated.	3·6	5·4	
Three negative results in five stools.	Observed.	1·0	9·0	10
	Calculated.	3·0	7·0	

(*e*) A few patients were observed over periods of more than six days. (i) Thirty-eight patients were observed for nine days; (ii) 29 tuberculous patients, with no known previous history of dysentery, were observed for twelve days; (iii) 36 miscellaneous patients were found to be passing cysts only infrequently, and were observed on varying numbers of days (six to sixteen), not necessarily consecutive, but spread over periods from one to six months. The results are shown in Table VII.

Only one patient, in series (ii), gave positive results on all days; the other 35 passed cysts on only one, two or three days. For these, who gave two or three positive results, the number of negative observations between two successive positive results varied from 0 to 11, with a mean of 4·5.

TABLE VI.

Presence or Absence of Cysts.	Ancillary Characters.		Total.
	One or More Observed on One or More Days.	None Observed.	
Cysts observed on one or more days	44	424	468
No cyst observed ..	75	167	242
Figures expected if association was at random ..	78·5	389·5	Deviation =
	40·5	201·5	+34·5
Total	119	591	710

III: Theoretical Considerations.

The outstanding problem in the class of work here reported is: "How many stools should be examined before a patient is passed as clear of the disease?" Although it is usually asked thus, the question in this form is incomplete. To obtain a reasoned answer it should be put somewhat as follows: "How many specimens, selected according to an agreed method of sampling, must be examined in order to determine with a stated probability that a patient's residual infection may be not greater than a stated intensity?" In the absence of more direct observation the criterion of infection will presumably be the average number of cysts passed per day.

Interpretation will in general be simplest when the data have been derived from strictly random samples. Two aspects of sampling have to be considered. The first is the frequency of the stools to be examined. If one wished to estimate the number of cysts being passed in a specified short period, such as a week, there would, of course, be no objection to the examination of every stool passed in that period. But that is not usually the situation. Rather one wishes to estimate the production which may be expected over a prolonged period, such as six months or a year. In view of the data presented in Section II (*c*), one week cannot be regarded as a random sample of a prolonged period. According to established principles of sampling, if, say, six stools are to be observed as representative of those passed in six weeks, the six days to be observed would be determined by random selection (by the drawing of lots, or from a table of random numbers *et cetera*). Alternatively, one might observe one stool per week, the particular day in each separate week being determined by lot; this might give a more accurately representative sample, but would introduce certain complications if estimates of variability and error were to be made. However, for practical convenience (for example, in directing an out-patient to come or to send specimens to the laboratory on specified days), many persons will prefer to ask for specimens at regular intervals, and in view of the findings in the second part of Section II (*c*), it may be anticipated that bias thus introduced should be trivial. (At the same time, strictly random sampling is not, in fact, so difficult to organize as inexperienced persons usually imagine.) But whatever properly considered method is chosen, it is to be noted for purposes of the following discussion that, although emission of cysts on successive days is not at random, randomness can be imposed on the days selected for observation.

The other aspect, the sampling of individual stools, is rather more complex. We may assume that cysts derived from ulcers high up in the colon are thoroughly mixed throughout the fæces; but those arising at the lower end, especially in the sigmoid, are likely to be more concentrated in the outer layer. This feature has not yet been adequately investigated; but the technique described in Section I above is probably satisfactory. Such observations as we have made, albeit too meagre to be worth presenting, suggest that there is little to be gained by observing more than one preparation from each stool, because the variation between stools is much greater than that between preparations from the same stool. (Random sampling of individual stools can, of course, be obtained by adding water to a suitable consistence, thoroughly mixing and taking a measured proportion. Such a procedure must be adopted if it is required to estimate the intensity of infection, but for mere detection it is not worth while.)

On the hypothesis that samples are random and properly representative of variation within stools (a questionable assumption), and if it is assumed that a man passes on the

TABLE VII.

Group of Patients.	Cysts First Observed in Stool: Number of Days.											Total.		Grand Total.
	1	2	3	4	5	6	7	8	9	14	15	Positive.	Negative.	
(i)	—	2	—	—	2	—	2	1	—	—	—	7	31	38
(ii)	2	1	—	—	2	—	—	—	1	—	—	6	23	29
(iii)	4	—	5	2	1	3	3	2	1	1	1	23	—	—
Total ..	6	3	5	2	5	3	5	3	2	1	1	36	—	—

TABLE VIII.

$\frac{k}{V}$	1	2	4	6	8	10	20	40	60
10^3 ..	0·003	0·007	0·013	0·020	0·026	0·033	0·064	0·125	0·181
10^4 ..	0·003	0·064	0·125	0·181	0·234	0·284	0·487	0·736	0·865
10^5 ..	0·284	0·487	0·736	0·865	0·931	0·964	0·999	—	—
10^6 ..	0·964	0·999	—	—	—	—	—	—	—

average 300 millilitres of fæces per day, of which one cubic millimetre is microscopically examined, then the probability of detecting a single cyst at any one examination is as follows: $p = 0{\cdot}001/300 = 0{\cdot}3 \times 10^{-5}$. Suppose further that the patient passes r_i cysts on the ith day of observation; $i = 1, 2, \ldots k$; then the probability of failing to observe at least one cyst in k specimens is as follows: $Q = 0{\cdot}999996^{(n_1 + n_2 + \ldots k)} \rightarrow 0{\cdot}999996^{kv}$, where v is the average number of cysts per day passed by the patient; and the probability of observing one or more cysts in k specimens is as follows: $P = (1 - Q)$. Table VIII shows P for $k = 1$ to 60, and for $V = 1{,}000$ to 1,000,000.

For small values of P, say less than 0·1, the change for a variation in the proportion of fæces examined from, say, one-third to two times the above proportion—that is, for $p^1 = 0{\cdot}1 \times 10^{-5}$ to $0{\cdot}7 \times 10^{-5}$—the revised value of P may be taken to a first approximation as $P^1 = P_p{}^1/_p$; but for P greater than 0·1, or for greater variations of the proportions sampled, revised calculations from the above general formulæ would be advisable.

It follows from Table VIII that to obtain a 50% probability of observing at least one cyst it would be necessary, by means of the methods outlined above: (i) from a patient passing an average of 1,000 cysts per day, to observe about 220 stools; (ii) from a patient passing an average of 10,000 cysts per day, to observe about 22 stools; (iii) from a patient passing an average of 100,000 cysts per day, to observe about two or three stools. From a patient passing on an average 1,000,000 cysts per day, examination of two stools may be expected to give almost certain detection, the probability of failure being only 0·001 (or in other words, one chance in a thousand).

It has been pointed out above that the sampling of stools can be arranged to conform with the hypothesis underlying this table. The sampling within individual stools may not so conform; inasmuch as the technique described above may be expected to err on the side of increasing the probability of detection, as compared to a strictly random sample, the table may be supposed to show lower limits for the probabilities of detecting given infection intensities with varying numbers of specimens.

Consideration has been given to the possibility of empirically estimating from the data the proportion of carriers undetected in varying numbers of examinations. All such attempts must, however, involve extrapolation, and it seems quite impossible to derive any theoretically valid curve which could give estimates sufficiently dependable for serious consideration.

Summary.

The paper describes observations made at Nakompaton prisoner-of-war hospital in Thailand on the frequency of observation of cysts of *Entamœba histolytica* in stools from patients convalescent after amœbic dysentery.

Table III in Section II (*b*) shows the numbers of cyst-passers detected on each of six successive days when stools were examined. There appears to be no way of deducing from these data how many carriers would be missed in an examination of any given extent. Observations on successive days are correlated, so that an examination covering only two or three successive days cannot be considered as representative of the patient's average cyst production.

In Section III principles of sampling are discussed, and it is shown how, if random sampling is assumed, the probability of detection may be estimated for varying sizes of sample taken from patients with varying intensities of infection.

Acknowledgements.

The observations were made by Major A. T. H. Marsden, Royal Army Medical Corps, and myself. Our thanks are due to L. R. Davidson and F. E. Drion for assistance in preparing specimens and recording the data.

Reviews.

PROGRESS IN NEUROLOGY, PSYCHIATRY AND ENDOCRINOLOGY.

THE publishers of the practical medicine series of year books combine neurology, psychiatry and endocrinology in one volume. The volume for 1945 dealing with these subjects is a worthy successor to those of previous years.[1]

In the preface of the part dealing with neurology, the editors, Hans H. Reese and Mabel G. Masten, discuss the scope of a neurologist. They hold that a neurologist is a student of the development, integrated functions and disintegrated pathology of the nervous system, who after acquiring a sound general medical education and a background of science has become a physician. A neurologist, they state, must remain abreast of the literature and of the research achievements in anatomy, physiology, biochemistry and pathology; he must be familiar with neurosurgical advances. He must be able to evaluate human relations, behaviour and the principles of dynamic psychology; he must apply psychotherapeutic procedures, without which no physician can practise medicine. There are few sharp borders to be drawn between the closely related clinical fields of neurology, psychiatry and surgery, at least in practice. A neurologist diagnosing conversion phenomena must be able to explain the underlying mechanisms, just as he must differentiate and coordinate psychotic reaction types in organic cerebral lesions. The sections of the part of the work devoted to neurology are four in number: (*a*) anatomy, physiology and pathology; (*b*) epilepsies; (*c*) disorders of the central nervous system—the abstracts here are subdivided into groups according to certain types of lesion, or structure affected; (*d*) diagnostic procedures. The first thing that strikes the reader is that the abstracts are remarkably full, several pages sometimes being devoted to one. The blood supply of the nerves of the upper limb in man have been described by Sydney Sunderland, of Melbourne. The subsection on head injuries is valuable. There is a discussion on neonatal asphyxia, its encephalic residuals and the mechanism of their production. Other subjects mentioned include the disability arising from closed head injury, chronic post-traumatic head symptoms, mental symptoms following head injury and pathological changes following brain trauma. In the subsection devoted to cerebral infections many diverse subjects are included, for example, cerebral symptoms in malaria, torula infection of the central nervous system, cysticercosis, tetanus, penicillin in the treatment of brain abscess, and porphyria in relation to nervous and mental disease. In another subsection recovery from tuberculous meningitis is recorded. A long reference is made to pressure effects associated with cervical and with rudimentary and "normal" first ribs. Amimia or emotional paralysis of the face is mentioned, and a reference to "restless legs" is included.

The part on psychiatry is edited by Nolan D. C. Lewis. In his preface he refers to the influence of modern psychiatry on all branches of medicine and surgery. The sections deal with general topics, child psychiatry, schizophrenic psychoses, organic reactions, toxic psychoses, psychoneuroses and psychosomatic disorders, and military psychiatry. In the subsection on organic reactions some interesting photomicrographs are included.

The part on endocrinology is edited by E. L. Sevringhaus, who states that the question whether to relate the endocrine system to the nervous system or to nutrition can be

[1] "The 1945 Year Book of Neurology, Psychiatry and Endocrinology"; Neurology—edited by Hans H. Reese, M.D., and Mabel G. Masten, M.D.; Psychiatry—edited by Nolan D. C. Lewis, M.D.; Endocrinology—edited by Elmer L. Sevringhaus, M.D.; 1946. Chicago: The Year Book Publishers Incorporated. 7" × 4¾", pp. 720, with illustrations. Price: 24s.

answered only by the statement that both relationships are important, but that the endocrine organs are really related to the entire body and to virtually all physiology. The part is divided into two main sections dealing respectively with the pituitary group and a miscellaneous group of glands. The first of these is subdivided into matters related to the "posterior pituitary", the "intermediate pituitary" and the "anterior lobe"; finally there are "miscellaneous pituitary matters". The section on the miscellaneous group of glands deals with the thymus, the spleen and antihormones. In the pages devoted to the thyreoid a great deal of useful information is to be found. The drug thiouracil is discussed from several points of view. The adrenal cortex is another subject that is dealt with at some length and in an efficient way.

A YEAR BOOK OF GENERAL MEDICINE.

"THE 1945 YEAR BOOK OF GENERAL MEDICINE" is divided into five parts, each of which has its own editor or editors.[1] It is one of the twelve volumes comprising the "Practical Medicine Series" of year books founded in 1900 and published continuously since then. The series is well known and needs no introduction to Australian practitioners of medicine. The articles chosen for inclusion in the year book come from journals in many different countries. Clearly the work included is not always entirely new or epoch-making—it would be disconcerting to find that the new work each year always filled a volume of approximately the same size. The work that is included, however, nearly always throws fresh light on a subject; sometimes the illumination comes from an apt remark by the editor, printed in small type at the end of an abstract.

The first part of the volume under review deals with infectious diseases and is edited by George F. Dick. The first reference is to work by an author who holds that the common cold is not caused by a virus but by a hæmolytic streptococcus. The editor properly remarks that in all probability the colds described are exacerbations of chronic nasal infection, and not colds at all. Among the other conditions discussed are dengue, hepatitis, influenza, malaria, poliomyelitis, rickettsial and virus diseases, streptococcal infections, tetanus and so on. The most important section of this part of the work is devoted to a series of articles on penicillin—its action on bacteria, methods for its estimation in blood serum and other body fluids, its concentration in blood serum after various doses given by various routes, its oral administration in oil, the enhancement of its concentration in plasma of dogs by the simultaneous administration of para-aminohippuric acid, the synergic action of penicillin and the sulphonamides, accounts of results obtained in a wide variety of infections by its use.

The part on diseases of the chest has been edited by J. Burns Amberson. In the section on function reference is made to an important article on bronchospirometry, a method by which the functions and volumes of each lung are determined separately and simultaneously. The work presented is by Pinner, Leiner and Zavod, of New York, who have attempted bronchospirometry 380 times and have obtained 270 satisfactory records. They hold that bronchospirometry is indicated whenever an irreversible operation on one lung is contemplated, in order to determine the functional capacity of the contralateral lung. Pathogenesis of pulmonary atelectasis has been discussed by R. F. Vaccarezza. Hæmothorax has been dealt with in articles by three authors or groups of authors and reference is made to the treatment of *empyema thoracis* with penicillin. In connexion with the latter the editor refers to the possibility of the creation of a permanent dead space in the pleura. A conservative view of aluminium therapy in silicosis is presented in the light of animal experiments by L. U. Gardner *et alii*. The use of penicillin in the treatment of intractable asthma is mentioned, and a useful statement on the surgical treatment of primary carcinoma of the lung is given. Twenty-eight pages are devoted to discussions on different aspects of pulmonary tuberculosis.

The part on diseases of the blood and blood-forming organs and on diseases of the kidney is edited by G. R. Minot and W. B. Castle, and covers 160 pages. The editors introduce the section on transfusion of blood and blood substitutes with an article summarizing the work of E. J. Cohn and his associates on the chemical fractionation of human plasma in connexion with a search for improved blood substitutes. They include in this section many articles dealing with Rh factors; as they remark, these give the reader a good idea of the present status of the subject. The different types of anæmia are discussed, and reference is made to the use of radioactive phosphorus in the treatment of *polycythæmia vera*. The section on agranulocytosis includes two references to this condition following the administration of thiouracil. The section on diseases of the kidney contains reference to the physiology, pathology and clinical aspects of renal disease.

The part on diseases of the heart and blood vessels is edited by William D. Stroud. The first three sections of this part deal with ætiological diagnosis, anatomical diagnosis and physiological diagnosis. In the first reference is made to such subjects as atherosclerosis, Mönckeberg's sclerosis, bacterial infection, congenital anomalies, effort syndrome, hypertension, hyperthyreoidism, hypothyreoidism, psychoneuroses, rheumatic fever, syphilis and trauma. Ligation of the *ductus arteriosus* is mentioned, and the editor states his impression that more patients are being operated on for this condition than is justifiable. Under anatomical diagnosis are included thrombosis of the coronary arteries and pericarditis; under physiological diagnosis the anginal syndrome is mentioned. Sections on electrocardiography, treatment, miscellaneous subjects and the peripheral blood vessels complete this part of the book.

The last part, on diseases of the digestive system and metabolism, is edited by G. B. Eusterman. Sections are devoted to diseases of the œsophagus and stomach, the biliary tract and pancreatic system, diseases of the small bowel and diseases of metabolism and nutrition. The frequent association of dyspepsia with active service conditions is shown by reference to an article on civilian dyspepsia. The articles on peptic ulcer in the army are full of interest. Large melena of obscure origin is discussed and mention is made of vagotomy in the treatment of peptic ulcer. An article on the conservative management of acute pancreatitis is worthy of special mention. Under the heading of diseases of metabolism and nutrition reference is made to many articles dealing with the results of vitamin deficiencies.

[1] "The 1945 Year Book of General Medicine", edited by George F. Dick, M.D., J. Burns Amberson, M.D., George R. Minot, M.D., S.D., F.R.C.P. (Edinburgh and London), William B. Castle, M.D., S.M., M.D. (Hon., Utrecht), William D. Stroud, M.D., and George B. Eusterman, M.D.; 1945. Chicago: The Year Book Publishers Incorporated; Melbourne: W. Ramsay (Surgical) Proprietary Limited. 7" x 4¾", pp. 768, with illustrations. Price: 24s., post paid.

SEX HORMONES.

THE text of Dr. H. Burrows's "Biological Actions of the Sex Hormones" is in some respects unsatisfactory after the expectations aroused by the preface.[1] The book was designed in part to keep those occupied with other matters informed of the rapidly growing literature on sex hormones as well as to provide a reliable foundation for further research. The sections of the book are devoted to gonadotrophins, gonadal hormones, androgens, œstrogens, progestins and sex hormones of the adrenal cortex. There is also an appendix providing details of proprietary sex hormone preparations and synonyms.

As a guide for those engaged in other lines of research, the chief defect of the book is a tendency of the author to summarize findings badly and not to evaluate them. The arrangement of the sections is at times a little startling; their inactivation, for example, being the work first considered in the section on gonadotrophins.

For those working on sex hormone pharmacology and biology, the detailed compilation of data culled from a formidable range of publications will be of inestimable value. But here again serious defects occur, such as a detailed discussion on the separate identity of the interstitial cell stimulating hormone and the follicle stimulating hormone, from which the all-important work of the laboratories of Fraenkel-Conrat and Van Dyke is omitted. These workers and their colleagues prepared in 1940 chemically pure interstitial cell stimulating hormone and biologically pure follicle stimulating hormone.

The author has been associated mainly with the Chester Beatty Research Institute of the Royal Cancer Hospital, and the relations of the hormones to new growths are considered in some detail. In regard to chemical structures of the hormones, where these are known, a summary of the relevant information is given, along with references to sources of detailed chemical information. There is a very extensive bibliography and a good index. The book is very well printed and arranged.

[1] "Biological Actions of Sex Hormones", by Harold Burrows, C.B.E., Ph.D., F.R.C.S.; 1945. Cambridge: Cambridge University Press. 9¾" x 6¼", pp. 524. Price: 42s. net.

The Medical Journal of Australia

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THE WORLD FOOD SHORTAGE.

As long ago as 1943 great concern was felt for the conditions which had to be faced by the peoples of the occupied countries of Europe in the matter of food. The situation at that time was discussed in these pages in the issue of December 4, 1943, in the light of a book, "Starvation in Europe", by Geoffrey H. Bourne. In the issue of December 18, 1943, it was possible to discuss the decisions reached at the United Nations Conference on Food and Agriculture, which had been held at Hot Springs, Virginia, in May and June of that year. At that conference it was pointed out that there had never been enough food for the health of the people of the world and that considerable expansion of food production would be necessary. In addition to the problem of the production of food, there would be the problem of its distribution. No less than forty-four governments and authorities were represented at the conference. Certain declarations, recommendations and resolutions were adopted, and these were directed towards increased production and cooperation in the distribution of food. Since 1943 the food position has deteriorated and the world is faced with a food problem of the greatest magnitude. It has been stated authoritatively that, without full and successful planning, more people may die from starvation before the end of 1946 than died from causes of war between the years 1939 and 1945. The urgency of the position is not generally recognized. Not so long ago it was pointed out in *The Times* that there has been much careless talk, even in high circles, about the "ninety days' crisis", and many people, particularly in America, believed that once the world had passed through the ninety-day period increasing supplies would be available and famine banished from the earth. "It would be difficult", we are told, "to find a statement further from the truth." The shortage of food throughout the world is certain to remain critical for four or five years.

This is not a matter that concerns certain parts of the world only. Australia has had a privileged position in the matter of food. It is true that large amounts of food have been sent from the Commonwealth to other, less fortunate, parts of the world. At the same time the question may well be asked whether most of our people understand the gravity of the situation and also whether more could not be done to send help to stricken parts of the world. In Great Britain His Majesty's Government has been concerned with the need for wide understanding of the position, because the people of the Home Land, already tried almost to the limit of their endurance, have been asked to make still further sacrifices for the suffering peoples of other countries. The Minister of Food has prepared a White Paper which was presented to Parliament in April, 1946, and has been printed by His Majesty's Stationery Office. The paper is published to set out the facts of the present crisis, to show how the crisis developed, and to trace the steps taken by the Government in the face of it. It will be well to bring to the notice of Australians the first two aspects of this White Paper.

Long before the end of the war it was foreseen that the shortage of fats, meat, dairy products and sugar which had developed during the war would remain for a considerable time after it was over. Towards the end of 1945, however, it became apparent that supplies of grain would be seriously inadequate and that it would no longer be possible to make up for deficiencies in other foods by increased consumption of cereals—the problem was no longer one of austerity, but of absolute shortage. This state of affairs arose largely from an exceptional succession of droughts in many of the world's main producing areas, coming on top of the already serious dislocation of the world's agricultural economy and trade during six years of war. The following figures are illuminating. Before the war the annual production of bread grains (wheat and rye) in Continental Europe, excluding the U.S.S.R., was 56 million tons. During last autumn the production was only 31 million tons, and a year earlier the amount produced was 46 million tons. There was a proportionately heavy fall in the production of meat and other livestock foods. This meant that the stated import requirements for the continent of Europe for the year July, 1945, to July, 1946, amounted to 15·6 million tons as compared with a pre-war import of 3·7 million tons. Even if these quantities could be obtained they would be about 25% less than those of an average pre-war year. The other deficit countries (India, China, French North Africa, South Africa and certain others) present a similar picture. In the East there has been a serious fall in the production of rice in countries occupied by Japan. In Burma and Siam production in 1946 is estimated at only 4·9 million tons against a pre-war average of 8·4 million tons. Further, in the Far East the production of vegetable oils and sugar has fallen to such an extent that relief is needed in some countries which before the war used to export substantial quantities. In addition to droughts which are described as the culminating factor in the situation (the most severe droughts have been in Europe, French North Africa, the Union of South Africa, New Zealand and India) other factors are named. Among these, in addition to the actual devastation caused by war, is the dislocation of the world's transport system as a result of war. We also read that in Australia shortage of manpower has been an

important factor. Against the heavy losses there has been an increased food production in Great Britain, in the Middle East and in North America. The increase in Britain was small in relation to world totals, but this is not the case in North America, where an "impressive expansion" has been achieved. In the United States alone food production in 1945 increased by 25% over the average for the period 1935 to 1939. The opinion is expressed that the quantities of grain being fed to livestock are too high in view of the needs in deficit countries for grain for human food. Under conditions of scarcity the most economical use of grain is for the feeding of human beings direct and not for its conversion through animals into meat and eggs for human food. Normally three and a half pounds of grain and concentrated feeding-stuffs are required to produce one pound of beef, five and a half pounds are required to produce a pound of eggs, and six to seven and a half pounds to produce a pound of pig meat. The current consumption levels are set out in a table; the amounts are stated in Calories per day as at the beginning of 1946. For European countries the figures are unofficial and somewhat rough estimates made on as nearly as possible a comparable basis. The average current consumption level per head per day of the whole population in the United States, expressed in Calories, was 3,150 or 102% of the pre-war figure. For Canada the figures were 3,000 and 100%; for Australia they were 2,900 and 97%; for Denmark and Sweden they were 2,850 to 2,900 and 90% to 95%; for the United Kingdom they were 2,850 and 95%; for France, Belgium, Holland and Norway they were 2,300 to 2,500 and 75% to 80%; for UNRRA countries (Greece, Yugoslavia, Czechoslovakia and Italy) they were 1,800 to 2,200 and 70% to 75%; and for ex-enemy countries 1,600 to 1,800 and 50% to 60%. In certain European countries the lot of the farming population is much better than that of the non-farm population. Thus in the last three groups mentioned (France, Belgium, Holland and Norway; the UNRRA countries and the ex-enemy countries) the average Calorie consumption among members of the population who do not work on farms is stated respectively to be 1,800 to 2,300, 1,400 to 1,800 and 1,200 to 1,400. When an average is stated it is, of course, clear that many persons will have a consumption below the average. The table to which reference has been made does not deal with any of the Eastern countries. We can thus understand that Mr. Hoover spoke truly when he described (as reported in the newspapers) the "measure of hunger" of 800 million people, more than one-third of the world's population. About 100 million people, he said, would be reduced to a 2,000 Calorie level, 100 million would be reduced to a 1,800 Calorie level, 150 million to a 1,500 Calorie level, 150 million to a 1,200 Calorie level, and about 300 million or more would be reduced to a 900 Calorie level or lower. The last-mentioned level, of course, means slow starvation.

The world situation in regard to food is certainly grim. Naturally the reader will ask what sources of supply are available and how they can be brought to the place where they are needed most. The White Paper contains a table setting out the supplies of wheat in the four main exporting countries—United States, Canada, Argentina and Australia. The figures for Australia may be reproduced. The total average annual supply (opening stock *plus* the crop) for the pre-war years 1934-1935 to 1938-1939 was 5·9 million tons; for the period 1939-1944 the average total was 7·1 million tons; for 1944-1945 the total was 5·7 million tons, and for the year 1945-1946 it was 5·0 million tons. The figures for domestic use (food, seed, animal feeding and so on) for these four periods were respectively (in million tons) 1·4, 1·7, 3·0 and 2·2. The amounts exported in these periods were respectively 2·9, 1·6, 1·6 and 1·1 million tons; end stocks were 1·6, 3·8, 1·1 and 1·7 million tons. It is not necessary for our present purpose to discuss in detail the present position regarding bread grains, rice, livestock numbers and feeding-stuffs, meat, oils and fats and sugar. The fact to be remembered is that in regard to many of them no early relief from shortage is to be expected. If the situation is to be met in anything like a satisfactory way, international agreements based on a realization of national responsibility will have to be made and observed. The burden will necessarily fall on the more favourably situated countries, and Australia is one of them. The example of Great Britain is something of which the British race can be proud, but this pride is worth nothing if it begins and ends with words of admiration. If we admire, we must also act. Until Australia does all that lies in her power we have no right to be content. Production and distribution are the two objectives. It is no use to attend to one without the other. Political ideas or ideals should not be allowed to interfere with either objective; unfortunately at present they do. Australia should be large-minded enough to find a way out of any political impasse, especially when the well-being of other peoples is at stake. In the long run it is the individual in any nation who is responsible for its action. We can thus conclude with words that have been credited to Mr. Hoover:

> If we can succeed in persuading every man and woman, every nation, to do their utmost, we shall master this famine, and we shall save the lives of hundreds of millions from the greatest jeopardy in all the history of mankind, we shall have saved infinite suffering.

Current Comment.

LESSONS FROM SPINACH.

THE use of New Zealand spinach has become popular in the United States of America by reason of the facts that this plant grows better than other edible greens in hot summer weather, and further that it responds so quickly and effectively to nitrogenous fertilizers. That it has a pronounced green colour has led to some extravagant recommendation by many nutritional monitors. Now comes a dispassionate investigation conducted in the Department of Horticulture, Missouri, by S. H. Wittwer, which puts a very different complexion on this topic, and in fact relegates this vegetable to the limbo of rhubarb leaves and wood sorrel.[1] New Zealand spinach actually contains so much oxalic acid and soluble oxalates that not only is its own calcium inadequate to make these innocuous as neutral calcium oxalate, but it robs other foods and even the body of the consumer of their calcium. The same condition has been found to be present sometimes in ordinary spinach and beet greens.[2] This finding inevitably suggests the phytic acid of cereal pericarps, and it is to be hoped that nutritional investigators will come to some convincing decision on this vexed question, for widely divergent views are held, from the extreme attitude that phytic acid of bran and pollard behaves like oxalic acid producing an adverse calcium balance, to the view expressed recently

[1] *Journal of Nutrition*, January 10, 1946.
[2] See, for example, Fincke and Sherman, *The Journal of Biological Chemistry*, Volume CX, 1935, page 421.

by a prominent British medical journal that phytic acid is "a figment of the imagination". The Missouri analyses of vitamin *C* in New Zealand spinach showed that this varies from 21 to 30 milligrammes per 100 grammes of fresh material; ordinary spinach has 40 to 100 milligrammes, whilst kale, turnip greens and broccoli have 75 to 200 milligrammes. Mere greenness therefore cannot be taken as a criterion of the excellence of a vegetable from the standpoint of either minerals or vitamins acquired by the consumer. Another point which came out clearly in this investigation is that when the New Zealand spinach was made to grow with extra luxuriance through the application of nitrogenous fertilizers, the vitamin *C* content fell. This has been found to be the case with other edible plants.[1] This consideration may have far-reaching results in our evaluation of the nutritional standing of any vegetable product. It was certainly disconcerting when Winikoff, working in a laboratory and under a direction which ruled out the possibility of error, found that the breast milk of Melbourne women had an insufficiency of ascorbic acid.[2] One is tempted to surmise that we have here the old antagonism between quantity and quality in food production and that economic pressure has forced the farmer, market gardener and orchardist to think of yield rather than grade of excellence. It is generally admitted that during this century the potato has undergone steady deterioration in taste, though the return per acre has increased. If a corresponding decline in nutritional value were also discovered, we should not be surprised. In a similar manner the striving after large numbers of eggs per fowl per year has led to a falling off in the colour, texture and composition of the yolks. New Zealand spinach plays a very minor role in nutrition, but it offers us two excellent lessons, one that the vegetable which happens to be green may not be worthy of recommendation, indeed may be discarded as unwholesome; the other is that the perpetual quest after bigger yields can be, from the nutritional standpoint, a matter for regret.

MEDICAL EDUCATION AND MEDICAL PRACTICE IN GERMANY DURING THE WAR.

AN interesting and informative article has been written by Colonel Robert M. Zollinger, of the Medical Corps, Army of the United States,[3] dealing with medical education and practice in Germany during the war. Before the war medical science in Germany was always held to have attained a high standard, and many persons no doubt supposed that the German genius for organization would be reflected in medical education and practice during the progress of the war. Zollinger holds that German superiority in matters medical is a fallacy, and he also finds that it is doubtful whether any significant developments have taken place in recent years in that country either in medicine or in surgery. He bases his remarks on what he learned on recent visits to the clinics of professors of medicine and surgery in three prominent German medical schools. Whether information gained from these centres (which by the way are unnamed) may be taken as representative of the whole country is probably open to argument, but some of Zollinger's impressions are summarized here for what they are worth.

The professors who were interviewed agreed that the calibre of the students was much below the pre-war level. This was thought to be due to several factors. Most of the students came from the army, being released because they expressed a desire to study medicine; many of them were anxious only to avoid the hardships and dangers of combat. In most cases they had neither the background nor the aptitude for study in a medical school. These men were physically fit, they were part of the Student Combatant Corps, and were under the direction of an army officer in each school. They wore uniforms, and were committed to the army while at school and at the end of their studies. A second group consisted of soldiers discharged from the services because of general physical disability or for other reasons. The State paid for the tuition of both these groups. A third group comprised those initially unfit for military service and a few who were excused from it because they had shown a particular aptitude for the study of medicine. These students paid for their own tuition, which amounted to 300 or 400 marks a year, and for their living expenses which were about 120 marks a month. After their graduation they were assigned to civilian or static military hospitals; a select few were trained as specialists after graduation. The last-mentioned group comprised some women, but women were not welcomed as students by the professors. The main difference between the war-time and the pre-war selection of students was that during the war most students were admitted by an outside authority. It was thought that many students with an aptitude for the study of medicine had no opportunity to enter a medical school.

Undergraduate teaching seemed to have been marked by curtailment and interference. At the beginning of the war the total period of study was reduced from six years or twelve semesters to five years or ten semesters. Early in 1945 it was further reduced to four and a half years or nine semesters. But this was not the only reduction, for interruptions were associated with military training and Nazi party functions. At least one semester of the premedical years was spent by the student as a medical corps man at the front. During the three medical years the student had to spend approximately two semesters working in an army hospital at the front. Apparently there was no systematic arrangement for the assignment of these students to army service. As a result of this state of affairs a great deal of clinical teaching at the bedside was omitted. Zollinger remarks that in the circumstances no faculty could have juggled a curriculum to cover the needs of such a fluctuating student body. In most cases the time actually spent by the student at the university was only three and a half years. In addition to this curtailment of study which had a prejudicial influence on the quality of medical graduates, other factors operated—there were the general hardships of war and the frequent, at times almost daily, interruption from air raids.

The ordinary methods of post-graduate training in which professors had up to forty assistants undergoing training were naturally upset. The number was greatly reduced and an increasing number of women had to be accepted as assistants. Research by the professors had to be curtailed; there was a lack of assistance, clinics and laboratories were damaged by air raids, and there was a shortage of drugs and other supplies. Still, most professors tried to continue their researches, but many of them were unable to publish their findings because of shortage of paper or because of arbitrary discriminations against them by Nazi officials. Some of the professors expressed resentment at the occasional appointment by Nazis of inferior men to important posts at the head of departments. They also appreciated keenly the loss to the universities of some men because of racial discrimination.

Zollinger's observations on medical practice can be only briefly mentioned. Sulphonamide therapy was looked on as the greatest advance made during the war, but the sulphonamide drugs were more highly regarded by the physicians than by the surgeons. Apparently both in civilian practice and in the army the use of sulphonamides in wound therapy was optional. The treatment of chest wounds was neither standardized nor modern. No new instruments or modern devices were observed in any of the clinics visited. The operating theatres of the clinics did not contain a single modern machine for the induction of anæsthesia. Zollinger has much to observe about the effect of interference and regulations by the State on the selection and activities of students and teaching staff. He thinks that irreparable damage has been done to the medical profession in Germany. This is a gloomy view, and, we hope, incorrect.

[1] Wittwer, Schroeder and Albrect: *Soil Science*, Volume LIX, 1945, page 329.

[2] THE MEDICAL JOURNAL OF AUSTRALIA, February 16, 1946.

[3] *New England Journal of Medicine*, May 7, 1946.

Abstracts from Medical Literature.

PHYSIOLOGY.

Role of Proprioceptors in Shivering.

J. F. PERKINS, JUNIOR (*The American Journal of Physiology*, December, 1945), reports the results of experiments in which shivering movements in cats were recorded by means of two phonograph pickups connected to a two-channel ink-writing oscillograph. Simultaneous records of the tremor were made in normal and in deafferented hind limbs. The movements of the limbs during shivering were rhythmic on the normal side, always irregular on the deafferented side. A hemidecerebellate animal and two animals with severance of one dorsal column shivered normally on both sides. The rate of shivering in a normal muscle could be changed by 100% or more by varying the mechanical resonance of the moving muscle. This was accomplished by adjusting the position of a pair of weights mounted on a lever attached to the tendon, or by varying the tension of a rubber band against which the tendon pulled. Deafferented muscles did not shiver rhythmically even with this arrangement. It is concluded that the rhythmicity and rate of shivering are determined peripherally by a mechanism which involves the proprioceptors and resembles that of reflex clonus. The rate of shivering in the normal animal is probably close to the resonant frequency of the moving part. A pacemaker action of the shivering centre appears unlikely.

Age, Sex, Carbohydrate, Adrenal Cortex and other Factors in Anoxia.

S. W. BRITTON AND R. F. KLINE (*The American Journal of Physiology*, December, 1945) report that age, sex, species and environmental temperature are important factors in resistance to low barometric pressures. Rat fœtuses and immature pouch opossums are highly resistant to anoxia. Infant animals show sharp declines in resistibility over the first one or two weeks of life, then become actually less tolerant for a period than older or adult animals (cat, rat). The relatively large size of the adrenal glands may be a factor in the superior resistance of young animals. Correlated probably are the high liver glycogen levels in infant rats. When glycogen levels have declined at three to four weeks of age, and the adrenals have also regressed (relative to rat weight), resistance to anoxia is found reduced even below that of adults. Out of six different mammalian types studied, the American brown bat showed phenomenally high resistance to anoxia. In nearly 2,000 tests, adult female rats showed much longer anoxic survivals than males over the temperature range 16° to 33° C. In 282 other cases (eleven series), females were similarly predominant. The large size of the adrenal glands in females, due chiefly to cortical tissue, may be a factor in these results. In infant male and female rats, in which adrenal weights are approximately the same, no sex differences in tolerance were observed. Survival times under anoxia are inversely related to environmental temperature. For a 10° C. drop, survivals were increased about 50%. Glucose administration improves greatly the resistance of rats to low barometric pressures. Carbohydrate meals and especially glucose injections were effective. Fully fed animals did somewhat better, also, than fasted controls. Insulin markedly reduced the ability to withstand anoxia. Carbon dioxide and oxygen (15/85) breathing before exposure to anoxic conditions was particularly helpful to fully fed rats. In combination, glucose injection and carbon dioxide and oxygen prebreathing proved most useful in raising resistance. Carbon dioxide and oxygen tested separately were not significantly effective when used as preexposure measures. Serum potassium, sodium and chloride levels were not affected by anoxic exposure. Beside the above data, a considerable array of evidence on young and adult animal responses points to specific adrenal involvement in anoxia. Infant rats without adrenals were strikingly reduced in resistibility to levels indeed below those of normal adult animals. Experiments on adrenal hypertrophy and cortical extracts offered supporting evidence. Altogether the results strongly indicate that while other factors are involved, the adrenal cortex aids greatly in extending life under anoxic contingencies. This is probably effected especially through its carbohydrate-regulating mechanisms.

Pulse Reaction to Step-Up Exercise on Benches of Different Heights.

E. R. ELBEL AND E. L. GREEN (*The American Journal of Physiology*, February, 1946) state that in normal subjects following the step-up exercise, for periods of thirty or sixty seconds, on benches of twelve to twenty inches in height, the pulse rates taken for thirty seconds, beginning one minute after exercise, are practically the same for the various heights of the benches and the durations of exercise. The pulse rate for the thirty seconds immediately following the stepping-up exercise depends upon the height of the bench and the duration of the exercise. After thirty seconds of exercise, the average increment is 3·7 beats per minute greater for each additional two-inch increase in the height of the bench. After sixty seconds of exercise, the increment becomes 5·6 beats per minute greater for each additional two-inch increase. The height of the bench and the duration of the step-up exercise as used in this study do not significantly alter the pulse rate if the pulse record is taken one minute after exercise.

Vascular Responses of the Nasal Mucosa to Thermal Stimuli.

H. J. RALSTON AND W. J. KERR (*The American Journal of Physiology*, July, 1945) report the results of experiments on human subjects, some of whom were normal, whilst others were susceptible to common colds. Temperature measurements of the skin and of the mucosa of the anterior portion of the inferior turbinate were made with constantan-copper thermocouples. Measurement of changes in the volume of the nasal space were made with a balloon which in the deflated state was 4·8 centimetres long. After insertion, the balloon was inflated to a pressure of 20 millimetres of mercury. They state that there was a rough parallelism between changes in nasal and finger temperatures when the nasal temperature and volume changes in response to cutaneous chilling and warming were studied. Local chilling characteristically results in a drop of nasal temperature, with substantial return toward the initial level within a few minutes. Local warming, after a transitory reflex drop, is generally followed by a permanently elevated nasal temperature so long as the stimulus is being applied. No systematic differences between normal and hypersensitive subjects have been observed. Local chilling of a subject exposed to a high environmental temperature results in a pronounced nasal hyperæmia. With general cutaneous chilling and warming, four types of nasal response have been observed, designated hyperæmia, ischæmia, turgescence and release of turgescence. These responses, involving changes in both temperature and volume of the nasal mucosa, are readily duplicated elsewhere in the body.

Effect of Ambient Air Temperature and of Hand Temperature on Blood Flow in Hands.

C. R. SPEALMAN (*The American Journal of Physiology*, December, 1945) reports some blood flow measurements made on a group of men subjects eighteen to thirty-five years of age. The men were sitting in uncomfortably warm, comfortable, and uncomfortably cold environments with hands immersed in water at temperatures ranging from 2° to 35° C. Under comfortable environmental conditions, blood flow was less in moderately cold hands (0·9 millilitre per 100 millilitres per minute for hands in water at 15° C.) than in very cold hands (4·3 millilitres per 100 millilitres per minute for hands in water at 5° C.) or in warm hands (5·9 millilitres per 100 millilitres per minute for hands in water at 35° C.). A qualitatively similar relationship existed in uncomfortably warm and uncomfortably cold environments. In the uncomfortably warm environment, however, blood flow in moderately cold hands (water temperature, 15° C.) was about the same as in very cold hands (water temperature, 2° C.). At any given hand temperature, blood flow was greater the warmer the body. This effect was most pronounced in moderately cold hands (*circa* 15° C.) where blood flow in the case of the uncomfortably warm environment was about twenty times that in the uncomfortably cold environment, and least pronounced in very cold hands (*circa* 10° C.) where the difference was only about threefold.

The Action of Cardiac Ejection on Venous Return.

H. A. BLAIR AND A. M. WEDD (*The American Journal of Physiology*, February, 1946) discuss the mechanical relations between cardiac ejection, lung air pressure, chest wall movements and venous return. They state that the excess of arterial outflow over venous inflow to the chest during systole tends to create a partial vacuum of about 15 millimetres of mercury in the lungs. Collapse of the chest wall, however, permits only a

small fraction of this to be realized, about 0·5 millimetre of mercury according to derivations from measurements of transient increase of body volume with each heart beat. This partial vacuum will assist venous return, but it is not nearly as important as the negative intrapleural pressure in performing this function. The relative sizes of cardiac stroke volume and lung volume determine the partial vacuum which tends to be created in the lungs by the heart beat, and therefore the potential aspirating forces on venous blood and chest wall. The ratio of cardiac stroke volume to lung volume is approximately constant in warm-blooded animals. Consequently, the relative mechanical effects of ejection are similar in animals of all sizes. Decrease of lung volume caused by accumulation of blood in the chest and the accompanying changes of elasticity may be expected to increase the aspirating action of arterial ejection. That this occurs is not proven directly, but it is shown that the normal mechanical relations may be greatly altered in valvular disease of the heart. In advanced mitral stenosis, for example, the chest may expand rather than contract during systole.

BIOCHEMISTRY.

Alloxan.

R. S. TIPSON AND J. A. RUBEN (*Archives of Biochemistry*, October, 1945) have described two colour tests for use in the qualitative detection of alloxan (or its reduction products). These two tests have been applied to trichloracetic acid extracts of a variety of animal tissues and fluids. The results obtained suggest that alloxan (or its reduction products) may be a normal constituent of certain tissues. These observations may be interpreted as indicating that over-production of alloxan, or inability to destroy it, may result in a concentration of alloxan that is cytotoxic and eventuates in diabetes.

Biotin.

J. GARDNER *et alii* (*Archives of Biochemistry*, December, 1945) have carried out two studies of the biotin metabolism of ten college women who consumed a diet with a low, moderate and high biotin content and the moderate diet supplemented with 50γ and 100γ of biotin as the methyl ester. These diets contained from 9γ to 171γ of biotin daily. An increase of the biotin intake in food from 9γ to 33γ failed to cause a rise in the urinary biotin content, but the fæcal biotin increased 40%. When 171γ of dietary biotin were ingested, the urinary biotin rose markedly to more than five times that excreted on the low or moderate diet, while the fæcal biotin increased 80% over that of the diet with a low biotin content. Total biotin output averaged 9·0, 3·0 and 1·5 times the dietary intake for the diets with low, moderate and high biotin contents respectively. Thus, biotin synthesis was again indicated and relatively less synthesis seemed to take place with the diet with a high biotin content. When 50γ of biotin were added to the moderate biotin diet, a maximum of 30% of the additional biotin was accounted for in the urine. The maximum urinary output occurred on the third or fourth day after the beginning of the biotin feeding. A larger percentage of biotin was recovered when 100γ of biotin were ingested. A maximum of 72% was accounted for in the urine. The rise in excretion continued for the entire six days of higher intake. Fæcal biotin did not rise when either 50γ or 100γ of biotin were fed. The water-soluble biotin in dried fæces varied from 2% to 15% of the fæcal biotin. An increase in measurable biotin occurred when a water extract of fæces was acid hydrolysed. The diurnal variation in urinary biotin was studied in diets of low, moderate and high biotin intake. The hourly biotin excretion was greatest in the afternoon and evening and lowest during sleep and in the morning. Twenty-five or 50γ of biotin given with breakfast and dinner were excreted promptly, a large portion but not all of the additional biotin being found in the urine within six hours after ingestion.

Fatty Acids.

D. RITTENBERG AND K. BLOCH (*The Journal of Biological Chemistry*, October, 1945) have carried out experiments in which sodium acetate labelled with D and C^{13} was administered to mice and rats. The fatty acids and cholesterol were isolated separately from the livers and carcasses of the mice. Both lipids contained C^{13} and D, indicating the utilization of both carbon atoms of acetic acid in their formation. Degradation of the fatty acids suggests that the labelled atoms are distributed at alternate positions along the carbon chains. The finding that the cholesterol contains not only deuterium but also C^{13} is proof that both carbon atoms of acetate are utilized in the synthesis of cholesterol.

Hæmatopoiesis.

J. M. OSTEN *et alii* (*The Journal of Biological Chemistry*, October, 1945) have shown that purified human or beef globin fails to support growth and normal hæmoglobin formation in the rat. Supplementation of either type of globin with isoleucine, all other known dietary factors remaining unaltered, results in the maintenance of a normal concentration of hæmoglobin in the blood. The subsequent removal of isoleucine is followed by the development of a mild to severe anæmia and death of the animal. These observations indicate that isoleucine is needed for normal hæmatopoiesis in the rat.

Human Milk Fat.

J. B. BROWN AND B. M. ORIANS (*Archives of Biochemistry*, March, 1946) have examined a large specimen of human milk fat with reference to the amounts and nature of its component fatty acids. No evidence could be found for the presence of more than traces of acids below C_{16}. As a result of distillation of two specimens of methyl esters, prepared from this fat, and applying low temperature crystallization technique to the main fractions, a number of pure esters and acids have been isolated for the first time and characterized. The presence of mono-ethenoic acids from C_{10} to C_{18} has been verified; the C_{18} acids of this type were shown to be made up chiefly of oleic acid, together with isomeric octadecenoic acids. About 40% to 50% of the octadecadienoic acids was shown to be ordinary linoleic acid, the remainder being presumably the isolinoleic acid found in butter fat. Small amounts of both trienoic and tetrenoic acids of the C_{18} series were also shown to be present, although the amount is negligible. The finding of dienoic acids of series above C_{18}, reported by Baldwin and Longenecker, was confirmed, and in addition evidence was presented for the presence of high molecular weight trienoic acids. This specimen of fat is much more similar to human body fat than it is to a typical milk fat.

Anoxia.

E. L. HONE *et alii* (*Archives of Biochemistry*, December, 1945) have shown that natural mixed tocopherols given orally in physiological quantities to adult rats result in a time of survival to anoxic anoxia greater (+89%) than that of controls on a diet containing no vitamin *E* and with a low fat content. The effectiveness of tocopherol is decreased (to about +31%) when the diet contains fat at a 12% level. Fat in the diet decreases survival time of rats under conditions of anoxia, possibly by decreasing the animals' respiratory quotient. Injection of large single doses of δ-α-tocopheryl phosphate greatly increases the time of survival to anoxia of guinea-pigs on a "rabbit chow diet", but has no effect on rats.

Endocrine Tissues.

W. H. MCSHAN *et alii* (*Archives of Biochemistry*, January, 1946) have determined the succinic dehydrogenase activity of certain endocrine tissues, the uterus, seminal vesicle and prostate of the rat, and compared it with that of the liver. It was found that the adrenal gland has an activity of approximately half that of liver and approximately twice that of pituitary, uterus, thyreoid, thymus and testes. Pancreas, prostate and seminal vesicle tissues had little activity, the activity of the tissue of the seminal vesicle being least of all.

Thiamin Requirements.

C. A. MILLS *et alii* (*Archives of Biochemistry*, March, 1946) have shown that the thiamin requirement per gramme of food rises sharply with advancing age in rats adapted to tropical warmth or temperate coolness, while the absolute requirement in γ per day advances with increasing body size. A similar rise in dietary thiamin concentration was found necessary to prevent signs of acute polyneuritis and death with advancing age and increasing body weight. At eighteen months of age rats kept in tropical heat develop acute and fatal thiamin deficiency at dietary thiamin concentrations adequate to support optimal growth in temperate coolness. Thiamin requirement, therefore, seems largely conditioned by the mass of metabolizing tissue and relatively independent of caloric intake under conditions of voluntary *ad libitum* feeding. Protein and choline requirements per kilogram of food seem to change little with the reduced food intake per unit of body mass in advancing age. The conclusion seems justified that diets for the aged or those in ill health should be enriched with thiamin to compensate for reduction in amount of food consumed. It seems probable that the same principle should be applied with the sharp caloric restriction of weight-reducing and hot weather diets.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on April 26, 1946, at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, DR. A. C. THOMAS, the President, in the chair.

The Rehabilitation of War Neurotics.

DR. C. M. MCCARTHY read a paper entitled "The Rehabilitation of War Neurotics" (see page 910).

DR. H. H. WILLIS read a paper entitled "The Rehabilitation of War Neurotics" (see page 912).

DR. H. L. SPEARMAN said that the views of the opening speakers had been valuable and instructive, and he regretted that practising psychiatrists were conspicuous by their absence, although the Mental Hospitals Department of New South Wales was well represented. A committee dealing with psychiatric problems of the disabled soldier had been in existence for about two years, and had had many discussions. This probably accounted for the absence of practising psychiatrists from the meeting. Dr. Spearman then went on to refer to one or two points, chiefly arising out of Dr. Willis's remarks. First, he asked why there was such a shortage of psychiatrists, and what was the psychiatrist's point of view. It had taken him (Dr. Spearman) three years to gain his diploma in psychiatry, twelve months being spent in a mental hospital. Most men would not do that, and yet the diploma was a necessary qualification. Psychiatrists were not appreciated. Saint Vincent's Hospital had one psychiatrist; there he had had to conduct an out-patient clinic and deal with eighty patients per session. Sydney Hospital was in the same position. As far as Dr. Spearman knew, Prince Henry Hospital had no psychiatrist; the Reception House had no psychiatrist. How could people become psychiatrically minded if the big hospitals did not have psychiatrists? The Minister for Health said that he did not consider a psychiatrist necessary at the Reception House. Referring to Dr. Willis's remarks on peptic ulcer, Dr. Spearman said that Dr. Holmes à Court had laid it down that peptic ulcer occurred in those with a psychological turmoil; there had to be an anxious or nervous background. Speaking of facilitation, Dr. Spearman said that it did occur in private life. The patient might be cured, but almost always relapsed some time or other. Dr. Spearman could give no opinion on the question of facilitation among soldiers. Another problem was whether the neurotic tendency was hereditary or acquired. One authority stated that the neurotic was born, not made. Dr. Spearman did not think that that was the whole story; it was again the question of the soil and the seed, with preference to the seed. The cause of the trouble was usually to be found in the home life, and chiefly in the mother. Referring to Dr. Willis's remarks about pensions, Dr. Spearman said that he thought that nowadays everybody agreed that the man wedded to his pension never finally threw off his disability. Dr. Willis had said that he preferred diseases to wander. Dr. Spearman cited the type of patient who had suffered from headaches for years or all his life; if after treatment the headache moved to another spot, then the patient was told that it was sure to move out altogether. Dr. Spearman thought that one of the greatest disabilities was the fact that the patient in the country had to be brought to the city for advice. This was a pity; centres should be established in the country. He did not think it a good thing to group all neurotics in hospitals. The person with whom they were chiefly concerned was the man with the early neurosis—the type of person who suffered from pains here and there and was subjected to every possible test, no abnormality being found. It was no use having such a patient admitted to a big centre; the general practitioner could be of the greatest help in his case. Dr. Spearman had recently shocked a psychologist of high standing, who had referred a patient to him, by giving the patient a bottle of medicine; but Dr. Spearman was convinced that, as well as explanation of his condition, a bottle of medicine was essential for such patients. Referring to persons who never exceeded a mental age of twelve years, Dr. Spearman said that they never made anything more than bootmakers and farm-hands; they never progressed to any great heights or got a full education. With regard to the value of early diagnosis and treatment, Dr. Spearman commended the book entitled "The Nervous Soldier", by Professor John Bostock and Dr. S. Evan Jones, which had received considerable adverse criticism everywhere. However, people were beginning to realize that the description of the nervous soldier given in that book was sound. All journalists were neurotics. The family doctor failed to recognize early neuroses; he treated them as physical diseases, and such family doctors bred a lot of neuroses. When the patient ultimately reached the psychiatrist, his neurosis was very hard to uproot. Shock treatment was not good in all cases.

DR. A. T. EDWARDS said that he had rarely listened to two such diverse views. Dr. Willis had kept his feet on the ground and given a pessimistic account; Dr. McCarthy's paper was filled with youthful optimism. Dr. Edwards said that he himself still retained some optimism; but it was somewhat tempered by the general public's scornful attitude to minor psychoses. The general practitioners and the medical profession were leading the public in their scorn for neurotics. At the university, when the students were doing out-patient work, the physician examining a patient would say "functional", and pass him out. The place of psychiatrists in general hospitals suggested that this was still the attitude. Moreover, students were taught—and probably this went on still—that no case should be diagnosed as functional unless every avenue had been explored to prove that it was not organic. Dr. Edwards agreed with Dr. McCarthy that that procedure would lead to much more ill health and do more harm than to make a diagnosis dependent partly on the patient's personality. Dr. McCarthy blamed the psychiatrists for not making practical suggestions; but they had not been completely idle. Some two years earlier a meeting had been called and a scheme formulated for a psychiatric clinic had passed through one of the larger social agencies; this, it was hoped, would bear fruit before long, so that such a service would be established. Dr. Willis had mentioned social service workers and expressed a doubt of their value; Dr. Edwards thought that they could be very dangerous. Unless they were psychiatrically trained or were to be used only as almoners, it was better not to use them. Unless they were psychiatrically trained they had no knowledge of the psychodynamics of mental illness. The lay psychologist was coming into the picture because of the shortage of psychiatrists. Psychiatrists were so few that they were only half doing their job. Dr. Edwards agreed with Dr. Spearman on the question of the seed and the soil. The soil was probably important, but not so important as Dr. Willis would have them believe. This emphasis put on heredity was a challenge to the psychiatrist. If it was the cause of the minor psychoses, then psychotherapy was not likely to be at all effective. Yet he had seen dramatic recoveries occur, the patients remaining well for many years, where the only treatment had been psychotherapy. It was important not to believe that the underlying cause was an hereditary weakness in neuronic structure. If they believed that that was so, they had better give up psychiatry and take up eugenics.

DR. BASIL WILLIAMS, discussing the problem of the rehabilitation of servicemen, said that the problem was made large because the people were not examined properly when they were taken into the army. Very little provision was made for any examination, and many people were taken in who should not have been in the army. In any future war some scheme should be formulated to keep such people out of the army. The men who examined them were not qualified, not taught; some had been retired for many years and were out of touch with medicine altogether. Referring to pensions, Dr. Williams said that those who dealt with a lot of compensation cases realized the benefit of a lump sum payment. The whole problem could be minimized by this method and eventually much sickness and public money saved. The treatment meted out at the Randwick Military Hospital seemed to be approaching that at Sydney Hospital and at Saint Vincent's hospital—the clinics were hopelessly overcrowded, interviews were very short, the doctors were not trained in psychiatry or medical psychology, patients were given a bottle of medicine and a sedative and dismissed with a blessing. If there was some liaison with the general practitioner attending the patient, results would be very much better; but there was no such liaison. Dr. Williams said that he had written letters with several of his deserving patients, but had received no reply from the Repatriation Department. The patients had had very short interviews and had returned with bottles of medicine. Another particularly important problem was the rehabilitation of married couples. Sometimes the man had been married for many years and had been separated from his wife by a period of active service; he returned home and was given no guidance whatever. Some two years previously, during a post-graduate session, an excellent speech had been made by an American medical

officer; he had given information on how the American army treated its fliers when they were going down on leave. Some books had been prepared by the American army, and they could easily be copied by the Australian authorities. People who married during the war also had a great problem; they missed the usual flow of events (courtship, marriage, honeymoon, period of adjustment), and instead they suffered from a lack of ties, a lack of interest in building a home, and the necessity for living with parents or "in-laws", which never worked. Greater use could be made of general practitioners. Dr. Williams said that he felt strongly on the subject of medical education, and thought that at the present time the general practitioner was a very inadequate creature. But he was optimistic enough to think that there would be a return to the old-style general practitioner, who was respected and loved by everyone.

DR. E. B. TUNBRIDGE said that he had spent many years as a general practitioner in country districts, and during that period he was repatriation referee. There were two points that required comment. Dr. Spearman had said that the general practitioner lacked interest in the psychiatric problems of the patient. Dr. Tunbridge did not think that Dr. Spearman could have had much experience of country practice. There one lived with people and they grew up around one; it was essential to take an interest in the make-up of one's patients, and unless the country practitioner could diagnose and treat fairly well the ordinary neuroses he was unlikely to succeed in his district. To say that country practitioners were all lacking in interest was unfair. It was also unfair to say that the Repatriation Department referred patients back with a bottle of medicine and some phenobarbital tablets. Dr. Tunbridge had found the opposite in Western Australia. That liaison between the general practitioners and the Repatriation Department should be encouraged. A little more sympathetic understanding of everyone's problems, and of the general practitioner and his problems, too, would get them a good deal further.

Dr. McCarthy, in reply, said that he was surprised and pleased that Dr. Willis had agreed with him on so many points. Dr. Willis had been kind in refraining from criticism of the army psychiatric centre at Goulburn. The centre had done a very good job, and it was a tragedy that it should have been scattered to the four winds. They had got their patients well in an artificial environment. They were trying to get into touch with their patients by post. It was disappointing to find that many of them who were thought to be quite well were not readjusting themselves well in civilian life. Some contact with these people was necessary. Dr. McCarthy said that he was advocating that a central centre should be created, primarily for the ex-service neurotic, and later to be extended for the civilian neurotic. The whole problem centred round the time factor. Every worthwhile psychiatrist would allow at least an hour with his patient at the initial interview; it was necessary to give enough time on the first occasion to these people, who were really suffering. Two good books had been published recently on psychosomatic medicine. One of the advances in modern medicine was the discovery that accidents were not accidents, but an emotional escape from a difficult situation. It was significant that during the coming week Professor Lamble was to lecture on the physiology of the emotions. In advocating a better deal for the ex-service neurotic, Dr. McCarthy said that he thought the psychiatrist should be there, but in the background, for guidance and investigation.

Dr. Willis, in reply, said that he did not know why Dr. McCarthy should have been grateful for his lack of criticism; he was just as pleased to cooperate with Dr. McCarthy after his discharge as while he was in uniform. Dr. Spearman had spoken of the shortage of psychiatrists; Dr. Willis said that he deplored it. More especially did he deplore the fact that so many army-trained psychiatrists were giving up the speciality and going to other branches of medicine. That was probably because of poor remuneration. Dr. Willis said that Dr. Spearman was completely under a misapprehension when he said that he (Dr. Willis) disliked modern methods of treatment; on the contrary, they were very valuable, not in curing mental disorders, but in cutting short their course. If a schizophrenic could be kept working for five years by shock treatment, something useful to the patient and the community had been achieved. But Dr. Willis's point was that psychiatrists adopting physical methods were abandoning the older methods, for example, explanation and suggestion, which were the foundation of psychiatric treatments. He was hopeful that insulin coma would aid some of these people where shock treatment had failed to do so. Dr. Willis then referred to Dr. Spearman's statement that people with a mental age of twelve years were fit only to be bootmakers and farmhands; he asked what was wrong with bootmakers and farmhands? The whole population could not be psychiatrists. The rewards of bootmaking were not inconsiderable at the present time. Dr. Willis did not think that a person with a mental age of twelve years should be labelled mentally deficient. Dr. Edwards had referred to the role of heredity. Dr. Willis said that certainly, if one thought that heredity was the only factor in mental disease, one became fatalistic and not helpful to progress in psychiatric medicine. But still, some patients seemed to be born to go mad and were merely fulfilling their destiny by so doing. Dr. Willis did not say that that was always true, but it was wrong to deny the existence of an hereditary factor and always to ascribe the breakdown to environment of the pre-school years. Replying to Dr. Williams's remarks about the errors made in the examination of recruits for the army, Dr. Willis said that the same errors would be made in the next war as they were made in the former war. Each generation had its own war and would run it in its own way. There seemed to be natural errors which were repeated by each generation. But in the recently ended war a great deal of trouble had been taken to pick out in training camps recruits presenting the grosser forms of mental disorder. Those with minor forms could not have been picked out early. With regard to the complaint of lack of cooperation by out-patient clinics, Dr. Willis said that he was surprised to hear the charge against Randwick Repatriation Hospital. It was the desire of the authorities there to cooperate, and they would be only too pleased to receive any information from private practitioners and give them information for guidance after treatment. Dr. Willis went on to say that domestic unrest played a big part in the production of "delayed shell-shock". The problem of the wrecked home was an important one. He did not know how to cope with it; that was really a matter for chaplains. Finally Dr. Willis said that, although shock treatment might relieve mental symptoms, the disordered thinking remained; that could be changed only by the old hard way of psychotherapy.

Dr. Thomas, from the chair, thanked the speakers for the excellent papers and the discussion. He said that, as one who had practised general medicine for many years, he thought that the general practitioner must play a big part in the rehabilitation of the war neurotic. After all, in many cases he would be the local medical officer for the Repatriation Department. It was only as one grew older and one's practice became more interesting in a way, that one realized what could be done by practising psychotherapy. Even in his practice at the present time, which was not of a general nature, Dr. Thomas encountered many cases which twenty-five years earlier would have been difficult. People consulted him, thinking they were suffering from all sorts of vague conditions, mainly surgical; but by a few pertinent questions it was possible to penetrate right down to the foundation of the trouble, and by a simple explanation to send the patient away much happier. Dr. Thomas thought that in the case of returned men, the general practitioner with the aid of the psychiatrist would be able to do a great deal of good. Dr. Thomas also came upon such cases in sitting on repatriation tribunals; he said that the tribunals did not always please Dr. Willis, because they sometimes had to raise the assessments. However, in the majority of cases the tribunals thought that the Repatriation Department was doing the proper thing by the men who came to them. Many of them felt that there was something wrong, because so many of these men had to go before them and were receiving pensions. However, at the present meeting some of these matters had been elucidated. The man should not be made to fit into the scheme, but the scheme should fit the individual. Dr. Thomas said that he had in mind two men who had been prisoners of war in Malaya for four years, and on their return to Australia were in a neurotic condition. They went to him for friendly advice. He advised them to go back to work as soon as they were out of the army. This they did, and their condition had greatly improved.

NOTICE.

THE General Secretary of the Federal Council of the British Medical Association in Australia has announced that the following medical practitioners have been released from full-time duty with His Majesty's Forces and have resumed civil practice as from the dates mentioned:

Dr. John J. Willson, 642, New Sandgate Road, Clayfield, N.2, Queensland (May 16, 1946).

Dr. B. N. Adsett, Sandgate Road, Nundah, N.E.3, Queensland (May 20, 1946).
Dr. A. J. Foote, Alexandra, Wickham Terrace, Brsbane, B.17 (May 25, 1946).
Dr. David F. Millar, Alexandra, Wickham Terrace, Brisbane, B.17 (June 1, 1946).
Dr. G. A. McLean, Alexandra, Wickham Terrace, Brisbane, B.17 (June 17, 1946).
Dr. J. M. Banks, 119, Marsden Street, Parramatta (July 1, 1946).
Dr. S. D. Allen, 215, Macquarie Street, Sydney (July 1, 1946).
Dr. D. B. Wightman, 16, Livingstone Road, Petersham (June 10, 1946).

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held at the Children's Hospital, Carlton, Melbourne, on May 8, 1946, DR. A. P. DERHAM, the newly elected President, in the chair. Part of this report appeared in the issue of June 22, 1946.

Basal Arachnoiditis.

DR. M. L. POWELL said that it was always interesting to see patients some years after a severe illness. The patient shown by him had previously been presented to the Pædiatric Society in October, 1939. She was admitted to the Children's Hospital in June, 1939, aged thirteen years, with a history of headache and vomiting for three months. These symptoms had been increasing in severity during the previous two weeks, and were accompanied by diplopia, staggering gait and head noises intensified with the heart beats. On examination she was highly intelligent, and an excellent witness. There was gross bilateral papilloedema, amounting to four diopters, with a macular fan in the right *fundus oculi*. The visual acuity was $^6/_6$ on the left side and $^6/_9$ on the right. There was slight peripheral constriction in both fields, with enlargement of the blind spot. Left rectus palsy was producing the diplopia. Nystagmus was present of a type associated with cerebellar lesions—when the patient looked to the right there was a rapid out swing with a slow return, of coarse phase; and when she looked to the left rapid and fine phase movements occurred indicating some right cerebellar dysfunction. The left arm tended to fall away slightly in the outstretched position, and the finger-nose-finger test on that side was not performed as well as on the right. The left biceps jerk and the right ankle jerk were somewhat depressed; otherwise the reflexes were normal. Auscultation and X-ray examination of the skull revealed no abnormality, and the results of the Wassermann test and the test for hydatid disease were negative. The pressure of the cerebro-spinal fluid was four hundred millimetres of water, and the fluid contained no cells and no increase in protein content. All these findings appeared to indicate a rapidly growing posterior fossa tumour. In view of the fact that her vision might fail, exploration of the cerebellum had been decided on. On June 9, 1939, an attempt was made to examine the ventricular contents, but the result was not very satisfactory owing to failure of the basal anæsthetic. The ventricles, however, did not appear unduly dilated. Five days later Dr. Whitaker had made a suboccipital approach to explore the cerebellum, preceding this with aspiration of the ventricle through the opening made five days before. The usual cross-bow incision was made, and the arch of the atlas nibbled away. The dura was incised, and immediately great herniation of the cerebellar lobes occurred. No evidence of tumour was found. With very great difficulty, and by considerable ablation of cerebellar tissues, the dura was brought together again and the incision closed. A hopeless prognosis was given, and all awaited what appeared to be the inevitable end. The next eight weeks provided a problem in control and management that was distressing in the extreme. From an intelligent, attractive, alert, bright girl the patient became an incontinent, dysarthric, manic-depressive, delusional, hallucinatory wreck of her former self, calling violently across the ward, laughing in a horrible mirthless fashion, semiconscious at times, maniacal at others, her voice having dropped in tone, until all wished her a speedy release. After about six weeks she developed generalized hirsutism, and this, with the lowered pitch of her voice, amenorrhœa and attacks of fairly typical sham rage, suggested the possibility of a Cushing's tumour; but in view of later evidence this was apparently due to hypothalamic dysfunction caused by the distortion in basal architecture due to the operation. About eight weeks after operation, when things were at their worst, the father had asked if deep X-ray therapy could be given. This request could not be refused, and arrangements were made for the patient to go to the Royal Melbourne Hospital. Scarlet fever developed in the ward, and she was not sent, which was fortunate, as her recovery might have been wrongly ascribed to radiation. Shortly after this episode, routine examination of her fundi showed a rather surprising decrease in the swelling of her disks, and it became obvious that her condition was starting to improve. She was still mentally unstable, with hallucinations and delusions; she was still irritable and unpredictable in temper, but the flap was not bulging, and in general there were undoubted signs of recovery. She started reading and doing fine work with her hands, and finally left the hospital three months after she had entered it. Dr. Powell said that he presented her now, seven years later, apparently in normal health. There was no abnormal finding apparent on examination of the central nervous system other than some slight constriction of the temporal fields, and the right optic disk was slightly paler than the left, but was not abnormal. Visual acuity was $^6/_6$ on both sides. There was no nystagmus or other evidence of cerebellar dysfunction. Three months after operation she had had her first menstrual period and synchronously all her excess hair fell out, taking a week for the epilation to be complete. She had carried on, all through the war, a job entailing fine vision and coordination—that of draughtmanship.

Dr. Powell said that they were thus confronted with a very typical cerebellar tumour syndrome, relieved by operation and reviewed after seven years, the patient being still perfectly well, doing work which required extreme delicacy of touch and vision. The answer must lie in an inflammatory rather than a neoplastic lesion, and in retrospect this case conformed to a localized arachnoiditis syndrome. Dr. Powell went on to say that as long ago as 1924 Gilbert Horrax, working with the Grand Master, Harvey Cushing, collected a series of thirty-three cases in which operation had been performed at the Peter Bent Brigham Hospital in Boston. These cases were extraordinarily similar to the one presented. Five of these patients died, two within a few days of operation and three two years later. In four cases necropsy was obtained, and all showed histologically inflammatory thickening of the arachnoid membrane forming the cerebellar and basal cisternæ. Twenty-eight patients survived and were well from one to nine years after operation, and in these cases there was disclosed at operation a greatly dilated posterior cistern with its thickened arachnoid membrane containing pent-up cerebro-spinal fluid under increased pressure. In some of these cases a piece of the arachnoid was removed for examination and showed microscopically the slight thickening that was evident to the naked eye. Spinal arachnoiditis and chiasmal arachnoiditis were illustrations of the same condition. Horrax had pointed out that there often was some arachnoid thickening over the posterior cistern in cases of frank cerebellar tumour, and that an arachnoid cyst overlying an acoustic tumour was quite common so that the operative appearance could be fallacious in this regard. In regard to ætiology, five of the cases followed closely upon *otitis media*, and it was reasonable to suppose that there had been some extension from this focus; and in regard to the other cases ætiological factors had been most vague, and such things as cranial trauma, infected contusions of the scalp, ocular and pharyngeal infections, syphilis and gonorrhœa had been mentioned as possible causes. Influenza and encephalitis had also been called into question, but evidence for all these was indefinite. Horrax, in summing up this experience, concluded with a statement which was remarkably applicable to the case presented when he said: "The group of cases here presented have been clinically indistinguishable from tumours of the cerebellum. Exploration of the suboccipital region was therefore the procedure of choice, and in the absence of tumour all that could be done was to open widely the arachnoid cisternæ that were presenting, thus evacuating their contents. In the large majority of patients this was sufficient to effect entire relief of the pressure symptoms, at least for the varying lengths of time that they have been followed. We have not employed lumbar puncture as a diagnostic measure, believing the risk of fatality from foraminal herniation of the cerebellum to be too great in patients with increased intracranial pressure."

Dr. Powell said that the patient presented actually had a more stormy post-operative course than most of the survivors in Horrax's series. The hypothalmic syndrome

which she developed was most distressing to her parents and others. However, it did appear that she had completely recovered, and he could only assume that she had had a localized cisternal arachnoiditis with cyst formation, the structure of which was destroyed by the operative interference. There was a lesson to be learned in this case. Things were not always what they seemed. There were some who said before operation: "What is the use? Here is our old enemy of childhood, the sinister medulloblastoma. Why subject her to operation, with its distressing sequelæ of a bulging flap, *hernia cerebri*, and final dissolution when she could attain the latter goal so much more simply?" Like the cerebral abscess, the intradural blood cyst or the cerebral hydatid, the possibility of an arachnoiditis did offer a ray of hope. It was their duty to grasp at straws in apparently hopeless cases. Dr. Whitaker had grasped this straw with a grip so firm that to this day it had never relaxed.

DR. J. G. WHITAKER said that he had performed few operations with such a charming result! He could well recall the greatly increased tension present at operation. As soon as the dura was opened, cerebellar tissue oozed out. The subsequently happy sequel was not anticipated.

(To be continued.)

Special Correspondence.

CANADA LETTER.

From OUR SPECIAL CORRESPONDENT.

THE negotiations between the Provinces and the Federal Government concerning taxing powers and social services, including health insurance, have broken down in a most disappointing manner, with the Premiers of Ontario and Quebec militantly opposing the centralization necessary if Canada is to have an overall and equalitarian system of social services. It is interesting to note that the Premier of Quebec was met by 10,000 supporters of his opposing tactics on his return to Quebec City. However, the remaining seven Provinces may conclude agreements with the Federal Government and proceed to health measures on a national scale.

The Canadian Army has recently released details of its random survey for tuberculosis among 5,000 men in service in Canada in the age group below thirty-five. All army categories were included, and all the men had been in service for at least eighteen months. Three active cases were found, and eight men were suspected of minimal tuberculosis and followed. None of these showed X-ray signs on joining up. The survey touched 34 men who had had fibrotic or semicalcified lesions on enlistment. Of these, 28 showed no change, and six showed further resolution or healing.

An intensive "school" for food handlers has been organized in Saskatchewan, the object of which is to go to all towns where food is dispensed commercially, and, in cooperation with the trades concerned, to teach the essentials of food sanitation. Hotels and restaurants have welcomed this joint effort, and it is hoped that higher standards of commercial kitchen cleanliness will result, once the personnel concerned are given a chance to learn the cardinal rules through films, lectures and demonstrations.

Medical education is again to the fore in Canada, with the announcement that the University of Saskatchewan has just begun a new $600,000 building for its medical college. The University of British Columbia has just had its Professor of Public Health, Dr. C. E. Dolman, make a survey of over forty medical schools in Canada and the United States of America. He has pointed out that Canada has only half as many taxpayers to support each of its ten medical schools as the United States of America have to support their seventy schools. The extent to which out-patient diagnostic clinics have become widely patronized, when located at teaching hospitals, is well demonstrated at Duke University, Durham, North Carolina, where the 50,000 out-patients who come in one year travel an average distance of eighty miles.

An important development in out-patient diagnostic service has recently occurred in New York, where the Vanderbilt Out-Patient Clinic of the Presbyterian Hospital (Columbia University) has been converted to a "group clinic" comprising six internists, two surgeons, and one each from gynæcology, urology, dermatology, ophthalmology, oto-laryngology, neurology and psychiatry. By pooling expert opinion on one day, rather than on widely separated days, it will be possible to assess 35 cases daily or 9,000 per year, an increase over present numbers. For teaching purposes it means that students can see their patients right through, rather than seeing a group of strange conditions in unrelated specialties as heretofore. This concentrated and consecutive study of a few cases at a time will make for tidy habits of thinking medically, and the average student will feel more like a participant in the serious business of medical diagnosis, and less like a bored spectator at a variety show. The cutting down of overhead through the economy of laboratory investigations and of readmission procedures will appeal to hospitals everywhere. This pioneering move in New York will undoubtedly affect Canadian medical methods, and may lead to a type of self-supporting group practice on a semi-private basis at our teaching hospitals. There is certainly a growing concern to cut down on the "paper war" in our clinics, now that veterans are trying to return to the practice of essential medicine.

Correspondence.

CONGENITAL FIBROCYSTIC DISEASE OF THE PANCREAS.

SIR: I have read with interest and pleasure Dr. Blaubaum's discussion of "Congenital Fibrocystic Disease of the Pancreas", but was surprised to find that he made no reference to Dr. Margaret Harper's contributions on this subject.

Dr. Blaubaum suggests that there was no clinical description of this syndrome before that given by Anderson in August, 1938.[1]

In 1930 Dr. Margaret Harper reported in THE MEDICAL JOURNAL OF AUSTRALIA[2] the details of two cases of "congenital steatorrhœa", defined the classical features of this condition and emphasized the distinction from cœliac disease.

Early in 1938, a few months before Dr. Anderson's article appeared in the *American Journal of Diseases of Children*, Dr. Harper published the details of eight cases of "Congenital Steatorrhœa due to Pancreatic Defect" in the *Archives of Disease in Childhood*.[3] This report included the results of post-mortem examination of four infants and a detailed review of the syndrome and its clinical features.

Yours, etc.,

June 18, 1946. LORIMER DODS.

References.

(1) D. H. Anderson: "Cystic Fibrosis of the Pancreas and its Relation to Cœliac Disease", *American Journal of Diseases of Children*, Volume LVI, August, 1938, page 334.

(2) M. H. Harper: "Two Cases of Congenital Pancreatic Steatorrhœa with Infantilism", THE MEDICAL JOURNAL OF AUSTRALIA, November 15, 1930, page 663.

(3) M. H. Harper: "Congenital Steatorrhœa due to Pancreatic Defect", *Archives of Disease in Childhood*, Volume XIII, 1938, page 45.

POLITICS AND THE MEDICAL PROFESSION.

SIR: Unfortunately Dr. Dane's letter of June 1, 1946, demands a reply, as it contains so many misleading statements, which I am quite sure Dr. Dane believes:

1. The phrase "from each according to his ability, to each according to his needs" ("needs", not "means", as misprinted, which is nonsense) has not been dropped by socialists, but is still their ultimate goal.

2. No country has yet fulfilled the first of the obvious requisites for its attainment, that is, enough goods and services to provide every member of the community with every need. Even America, before the war, was short of over 4,000,000 houses for her workers; and was so short of education at that time that she has now reaped the dreadful harvest of 10,000,000 illiterates. To take one need alone (I think the most important), there is not and never has been any country with enough suitable buildings, trained staff, and adequate equipment to give a full education, up to the highest standard, to every child capable of benefiting from it. My second requisite, or "mystic pomposity" if Dr. Dane prefers it so, was that some generations of reeducation in community service, instead of personal acquisitiveness, would be necessary before the great majority of the people were morally fit for such a system. I believe that with proper education and a decent social order human beings are capable of learning to behave reasonably to one another, which is all that would be necessary.

3. I did not attempt to discredit Dr. Dane's assertion that the birth of communism had no analogy with obstetrical practices. I only pointed out that his statement that there was never any violence in obstetrics was an example of the most extraordinary loose thinking about a branch of his own profession; I am grateful to him for supplying me in the present letter with other examples of deplorably loose thinking and reckless saying, because I wish to discredit him as a witness, and so help to discredit his most dangerous thesis.

4. "That communism and fascism are essentially the same." That seems to me dangerous, because we have just fought a most terrible war to destroy fascism; if communism is essentially the same the dreadful corollary inevitably follows that we must fight an even more terrible war to destroy it. Fundamental matters on which communism and fascism are diametrically opposed are: (*a*) Racial theory: fascism teaches that there is a "master race" to which all others are subservient, which lead to the horrors of anti-Semitism; communism teaches that all nations are equal, if given full opportunity to develop their latent powers; having acted on that teaching, the U.S.S.R. has no exploited race within its borders; to my knowledge even the most bitter Soviet-hater has not been able to point at one. (*b*) The declared aim of fascism is to create a master class ruling over a slave world; the aim of communism is a society in which there shall be no class divisions and every citizen shall have equal opportunity for full development. (*c*) Fascism restricts higher education to the dominant class; communism aims at giving the fullest education to all. (*d*) Fascism restricts woman to a subordinate position—to menial work, to the home, and to be available "for the recreation of the tired warrior"; communism affirms her complete equality (but not similarity) to man. (*e*) Fascism teaches that the end of man is to serve the State; communism that man should use the State to improve his individual lot, until he becomes so civilized that he no longer needs it. (*f*) The fascist glorifies war; the communist loathes it. I hope that when Dr. Dane considers this list of differences, which is by no means complete, he will not still think them "trifling details".

5. Without any attempt to learn the facts, Dr. Dane states that I am a communist, which is as false as so many of his other reckless statements. Fortunately for him I am a tolerant person, otherwise he would be defending a libel action.

Yours, etc.,

E. P. DARK.

Katoomba,
New South Wales,
June 10, 1946.

[This correspondence is now closed.—EDITOR.]

THE PLACING OF X-RAY FILMS IN ENVELOPES.

SIR: Whenever a series of a patient's X-ray films (say, of the chest) is being placed in an envelope, it is an excellent practice to see that they are arranged in chronological order with the most recent nearest to the flap of the envelope. I should like to commend this practice, which is not generally followed, to medical practitioners and radiologists. It saves time and trouble when the films are referred to subsequently; unless, of course, some careless person should disarrange them in the meantime.

Yours, etc.,

DOUGLAS ANDERSON.

185, Macquarie Street,
Sydney,
June 13, 1946.

MALARIA.

SIR: The letter in today's (June 1, 1946) journal from Dr. M. R. Finlayson and Dr. J. McF. Rossell, chief medical officers respectively of the New South Wales Government Railways and Department of Road Transport and Tramways, is timely, but I think tends to lay too much reliance on the blood slide "to clinch the diagnosis". Any physician with experience in treating malaria on active service in the tropics and in the returned serviceman at home will admit the existence of a clinical "apparent malaria" in which the blood slide often returns a "negative"—which after all is only a "not proven" verdict. Amongst other clinical signs tenderness over the spleen is a point of importance. Initial malaria appearing after return from the tropics is often due to the patient's carelessness in not keeping up the preventive "Atebrin" ingestion. Some cases appear particularly late after leaving the tropics. Some "certainties" give a negative and some "doubtfuls" give a positive. Some are not conventionally typical. Is it that "Atebrin" medication has produced a "groggy wog"?

The reference to the Repatriation Department is correct. Its efficiency in this matter is commendable. In response to a call the local medical officer attends, takes necessary drugs (quinine, "Atebrin" and "Plasmoquine"—11-day course for initial attacks, "Atebrin" 63-day course for recurrences), sends a blood slide by family messenger to Prince of Wales Hospital, Randwick (receiving a telephoned result same day), reports on Form 71 *re* the case and certifying probable incapacity for which sustenance is payable. One has never seen more than seven days needed nor requested. One agrees there may be a "malaria neurosis", but there is a possibility that the non-neurotic returned man often tries to battle through an unrecognized mild malaria.

Yours, etc.,

KEVIN BYRNE.

The Boulevarde,
Lakemba,
New South Wales.
June 1, 1946.

BACILLE CALMETTE GUÉRIN.

SIR: In your account of what seems to have been a most interesting address given by Dr. H. M. James in Melbourne, published in the current issue of the journal, reference is made to *Bacille Calmette Guérin*, and Dr. James is reported as saying (in reference to his own travels) that ". . . it is unstable and should be used within five days or at latest ten. It is made in Montreal, and it would be difficult to arrange supplies even with air transport". Presumably Dr. James is referring to transport to Australia from Canada.

My reason for writing is to inquire whether the same unfortunate outlook prevails in Canberra as previously. Exactly eleven years ago at the Pasteur Institute in Paris I personally received directly from Dr. Guérin himself a very generous offer to train any suitable Australian medical practitioner in the technique of the subculturing of "B.C.G." without cost, even in the matter of board and lodging; and further, Dr. Guérin guaranteed to supply on board the ship taking the trainee back to Australia at Marseilles, Toulon or Bombay, a thoroughly tested, efficient and innocuous culture of "B.C.G.", providing that facilities for subculturing by the trainee were provided on the ship. This generous offer was eventually declined by the "authorities" (if that is the correct word, Mr. Editor) in Canberra on the ground that it would not be safe to allow this strain of microorganism entrance to this country.

Possibly Dr. James is aware of a more scientifically sound outlook at present.

Yours, etc.,

J. A. R. MURPHY.

Waterfall Sanatorium,
Waterfall,
New South Wales.
June 17, 1946.

BLOOD LOSS IN CÆSAREAN SECTION.

SIR: Whilst very interested in Dr. Benson's article (THE MEDICAL JOURNAL OF AUSTRALIA, June 15, 1946), I would like to point out that the cases in the series are hardly comparable. First, in the selection of cases, the series delivered by Cæsarean section included cases of ruptured uterus, *placenta prævia* and preeclampsia. These conditions are apt to lead to a greater blood loss in themselves apart from any consideration of the method of delivery. The last-named condition especially, and the others to the extent of the increased blood loss, would tend to depress the plasma protein values. Secondly, Dr. Benson gives no information of the type of anæsthesia employed for the cases of Cæsarean section. Probably the majority of them had general anæsthetics, all of which, to a greater or lesser degree, would have a twofold effect; first, they decrease the contractility of the uterus, thereby increasing the blood loss, and secondly, they are toxic to the liver and would tend to depress the rate of recovery of the plasma protein. It would be interesting to see a similar set of results from a series of Cæsarean sections performed under regional analgesia.

Yours, etc.,

K. H. BROOME.

Coonamble,
New South Wales,
June 19, 1946.

Post-Graduate Work.

COURSES IN OBSTETRICS AND GYNÆCOLOGY AT BRISBANE.

THE University of Queensland Post-Graduate Medical Education Committee announces that the following facilities are available for post-graduate instruction in obstetrics and gynæcology:

1. A continuous series of three-weekly terms as supernumerary resident medical officer (or officers) at the Brisbane Women's Hospital under the supervision of the Professor of Obstetrics. Resident supernumeraries share in the work of the hospital. These facilities are intended for ex-service medical officers.

2. A series of lectures in obstetrics and gynæcology extending over two or three weeks as part of the second general revision course to commence shortly.

3. A series of tutorial classes in the relatively more advanced aspects of obstetrics and gynæcology, suitable for candidates intending to present themselves for membership of the Royal College of Obstetricians and Gynæcologists at the forthcoming examination in February, but also available to any practitioner desiring to attend. Tuition will be given in the relevant anatomy, embryology, physiology and endocrinology, and in the clinical, surgical and pathological aspects of the subject.

The fee for the tuition course will be £20, and classes will commence in July. Applications for enrolment, accompanied by a remittance for the amount of the fee, should be made to Dr. P. H. Macindoe, Secretary, the University of Queensland Post-Graduate Medical Education Committee, the Medical School, Herston Road, Fortitude Valley, Brisbane.

Obituary.

REDFORD JOHN WRIGHT-SMITH.

WE are indebted to Dr. Victor Hurley for the following appreciation of the late Dr. Redford John Wright-Smith.

Dr. Redford John Wright-Smith, who died suddenly in Melbourne on May 5, 1946, was born in 1896, and was at the height of his career as a pathologist and morbid anatomist at the time of his death.

After leaving Scotch College, Melbourne, he enlisted as a private in the First Australian Imperial Force and served overseas. At the termination of the war, while waiting to return to Australia, he commenced his medical course at King's College, London. On his return to Australia he completed his medical course at the Melbourne University, and after taking a high place in the final honours examination in 1924 he was appointed resident medical officer at the Royal Melbourne Hospital, and was a registrar the following year.

In 1927 he obtained the degree of M.D. (Melbourne), and directed his work chiefly towards pathology. He commenced his career as a pathologist first as a resident pathologist and later as non-resident pathologist at the hospital, working under Dr. Crawford Mollison, with whom he was very closely associated for the rest of his career.

In 1934 he went abroad and worked for some time with Professor H. M. Turnbull and Professor W. Bulloch at the London Hospital, and also with Professor Wood, of the Bernhard Bowen Institute of Pathology.

On his return to the Royal Melbourne Hospital he threw himself with enthusiasm into the work of building up and extending the pathological museum of the hospital. Specimens were selected and mounted especially for the purposes of clinical teaching, and accompanying each specimen were outlined the essential features of the clinical history of the case. He introduced several new departures, most of which were designed to correlate the clinical with the pathological work of the hospital. In this he achieved auspicious success, and the opportunities provided were largely used and appreciated by the staff and by the students.

In the new hospital at Parkville the increased accommodation and facilities provided for his department offered still further scope for his work, which he quickly took advantage of. The hospital has recently commemorated his work by naming after him the museum for which he was so largely responsible.

His weekly demonstrations of interesting specimens, with a running commentary on the clinical histories, were largely attended by members of the honorary and resident staffs, and became a feature of the work of the hospital. He was able to draw on an experience of more than 13,000 personally conducted autopsies of which detailed clinical pathological and histological observations had been carefully recorded. He continued also to maintain a close contact with clinical medicine. Because of his unique experience and ready recollection of the salient features of rare or unusual cases, his opinion was highly valued and frequently sought by his colleagues in difficult and obscure cases.

In addition to working under Dr. Mollison at the hospital, he also assisted him as coroner's surgeon and later succeeded him in this work in which he established a high reputation, both in the medical and in the legal professions, as an expert medical witness in forensic and medico-legal cases. He also succeeded Dr. Mollison in 1943 as Lecturer in Forensic Medicine at the Melbourne University.

He was a Fellow of the Royal Australasian College of Physicians, and also took an active part in the formation of the Medico-Legal Society of Victoria, of which he was one of the joint secretaries for several years. Much of the success of the society, in which opportunity is provided for the interchange of views between lawyers and doctors and for a better understanding of the problems in which they are mutually interested, is largely due to the energy and enthusiasm he put into this work.

He did a great deal of the editorial work in connexion with the publication of the *Proceedings* of the society, and he also acted in a similar capacity for *The Royal Melbourne Hospital Clinical Reports*.

With his many other activities during the war years he found time to do a good deal of work for the services, and held the rank of wing commander in his appointment as consultant pathologist to the medical services of the Royal Australian Air Force.

Among his outside interests he took a special interest, as an ex-serviceman of the 1914-1918 war, in the activities of the Legacy Club. He was a keen golfer and was a regular playing member of the Metropolitan Golf Club, where he played a more than usually good game the day before his death.

His sudden and unexpected death has deprived the Melbourne Medical School of an able and experienced worker whose place will be very difficult to fill.

The sympathy of his many friends is extended to his widow and four children in their great loss.

EUGEN HIRSCHFELD.

WE regret to announce the death of Dr. Eugen Hirschfeld, which occurred on June 18, 1946, at Brisbane.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Makinson, Jill, provisional registration, 1946 (Univ. Sydney), 45, Abbotsford Road, Homebush.

Johnson, Maurice Herbert, M.B., B.S., 1943 (Univ. Sydney), 49, New South Head Road, Vaucluse.

Marsden, Hugh Ernest, M.B., B.S., 1941 (Univ. Sydney), Prince Henry Hospital, Little Bay.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association:

Barbour, John Gordon, M.B., B.S., 1945 (Univ. Sydney), 6, Fitzwilliam Road, Vaucluse.

Bell, Betty Jeanette, M.B., B.S., 1945 (Univ. Sydney), 4, Gipps Street, Wollongong.

Chapman, Herbert Owen, M.B., Ch.M., 1907 (Univ. Sydney), 320, Piper Street, Bathurst.

d'Apice, Mary Dorothy, M.B., B.S., 1945 (Univ. Sydney), 28, Wolseley Road, Point Piper.

Davidson, Roger Madgwick, M.B., B.S., 1946 (Univ. Sydney), Marrickville District Hospital, Marrickville.

Flannery, Brian Peter, M.B., B.S., 1943 (Univ. Sydney), 28, Melrose Parade, Clovelly.

Giblin, William Eric, junior, M.B., B.S., 1945 (Univ. Sydney), Wahroonga Avenue, Wahroonga.

Hendry, Peter Ian Alexander, M.B., B.S., 1939 (Univ. Sydney), 16, Vernon Street, Strathfield.

Herlihy, William Francis, M.B., B.S., 1945 (Univ. Sydney), 138, Victoria Street, Ashfield.

Holland, Edward Henry, M.B., B.S., 1946 (Univ. Sydney), 98, Remly Street, Lakemba.
Hook, Reginald Harley, M.B., B.S., 1946 (Univ. Sydney), 19, Julia Street, Ashfield.
Hume, Ian Hamilton, M.B., B.S., 1945 (Univ. Sydney), 26, Woonona Avenue, Wahroonga.
Lane, Melva Joyce, M.B., B.S., 1945 (Univ. Sydney), 3, Chevron, 8, Dalhousie Street, Haberfield.
Nowland, Reginald John, M.B., B.S., 1945 (Univ. Sydney), 428, Darling Street, Balmain.
Phillips, Garth Jowett Blunden, M.B., B.S., 1942 (Univ. Sydney), Lake Crescent, Teralba, New South Wales.
Redshaw, Joan Margaret, M.B., B.S., 1944 (Univ. Sydney), 7, Gerald Avenue, Roseville.
Scobie, Roden Crichton, M.B., B.S., 1934 (Univ. Sydney), 201, Macquarie Street, Sydney.
Selby, George, provisional registration, 1946 (Univ. Sydney), 36, Dudley Road, Rose Bay.
Thomas, Ian Davies, provisional registration, 1946 (Univ. Sydney), 71, Woniora Road, Hurstville.

THE FEDERAL MEDICAL WAR RELIEF FUND.

THE following contributions to the Federal Medical War Relief Fund have been received:

Western Australia.

John Day, £10 10s.
H. E. H. Ferguson, £10.
J. C. Bennett, S. L. Harke, A. S. Johnson, W. S. Muir, W. P. White, W. H. Godby, £5 5s.
D. J. Oldmeadow, A. M. Clark, £3 3s.
Total: £58 6s.
Grand total: £12,492 15s. 8d.

Books Received.

"Lewis's 1844-1944: A Brief Account of a Century's Work"; 1945. London: H. K. Lewis and Company Limited. 8¼" × 6¼", pp. 98, with illustrations. Free on application.

"A Complete Outline of Fractures Including Fractures of the Skull", by J. Grant Bonnin, M.B., B.S. (Melbourne), F.R.C.S. (England); Second Edition; 1946. London: William Heinemann (Medical Books) Limited. 8¾" × 5½", pp. 672, with many illustrations. Price: 30s. net.

"Human Torulosis: A Clinical, Pathological and Microbiological Study with a Report of Thirteen Cases", by Leonard B. Cox, M.D. (Melbourne), M.R.C.P. (Edinburgh), F.R.A.C.P., and Jean C. Tolhurst, M.Sc. (Melbourne); 1946. Melbourne: Melbourne University Press. 9¾" × 7¼", pp. 161, with illustrations. Price: 25s.

"These Small Glories", by Jon Cleary; 1946. Sydney, London: Angus and Robertson Limited. 7¼" × 4¾", pp. 150. Price: 6s.

"The Principles of Anatomy: An Introduction to Human Biology", by A. A. Abbie, M.D., B.S., D.Sc. (Sydney), Ph.D. (London); Second Edition; 1946. Sydney, London: Angus and Robertson Limited. 8½" × 5½", pp. 374, with many illustrations. Price: 12s. 6d.

"Sir W. Arbuthnot Lane, Bart., C.B., M.S., F.R.C.S., His Life and Work", by W. E. Tanner, M.S., F.R.C.S.; 1946. London: Ballière, Tindall and Cox. 8¼" × 5½", pp. 202, with one photograph. Price: 15s.

"101 Clinical Demonstrations to Nurses", by Hamilton Bailey, F.R.C.S. (England); Second Edition; 1946. Edinburgh: E. and S. Livingstone Limited. 8¼" × 5½", pp. 136, with many illustrations, some coloured. Price: 10s. 6d.

Medical Appointments.

Dr. C. G. Thompson has been appointed Medical Referee, Launceston, Tasmania, in accordance with the provisions of Section 15 (2) of the *Workers' (Occupational Diseases) Relief Fund Act*, 1928.

Dr. I. H. Martin has been appointed Resident Medical Officer for Mental Institutions in the Hospitals Department, Adelaide.

Dr. C. Christie has been appointed Medical Officer of Health, Dumbleyung Road Board, Western Australia.

Dr. A. J. Beaumont has been appointed Medical Officer of Health, Municipality of Geraldton, Western Australia.

Dr. B. O. Bladen has been appointed Medical Officer of Health, Guildford Municipality, Western Australia.

Dr. J. A. Rolland and Dr. T. L. McLarty have been appointed members of the Board of Optical Registration, Adelaide.

Diary for the Month.

JULY 2.—New South Wales Branch, B.M.A.: Council Quarterly.
JULY 3.—Victorian Branch, B.M.A.: Branch.
JULY 3.—Western Australian Branch, B.M.A.: Council Meeting.
JULY 5.—Queensland Branch, B.M.A.: Branch Meeting.
JULY 9.—Tasmanian Branch, B.M.A.: Ordinary Meeting.
JULY 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JULY 9.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JULY 11.—South Australian Branch, B.M.A.: Council Meeting.
JULY 12.—Queensland Branch, B.M.A.: Council Meeting.
JULY 16.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JULY 17.—Western Australian Branch, B.M.A.: General Meeting.
JULY 18.—New South Wales Branch, B.M.A.: Clinical Meeting.
JULY 18.—Victorian Branch, B.M.A.: Executive Meeting.
JULY 23.—New South Wales Branch, B.M.A.: Ethics Committee.
JULY 24.—Victorian Branch, B.M.A.: Council Meeting.
JULY 25.—South Australian Branch, B.M.A.: Council Meeting.
JULY 25.—New South Wales Branch, B.M.A.: Branch Meeting.
JULY 26.—Queensland Branch, B.M.A.: Council Meeting.
AUG. 2.—Queensland Branch, B.M.A.: Branch Meeting.
AUG. 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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Medical Library

THE MEDICAL JOURNAL OF AUSTRALIA

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COMMONWEALTH OF AUSTRALIA. DEPARTMENT OF HEALTH.

PENICILLIN

(THE CALCIUM SALT OF PENICILLIN)

1 ampoule containing 100,000 Oxford units, 10/6.

This quantity is sufficient for a series of parenteral doses.
(Available also in boxes holding 5 ampoules.)

1 ampoule containing 15,000 Oxford units, 5/6

This quantity is intended for a single parenteral dose.
(Available also in boxes holding 6 ampoules.)

1 ampoule containing 5,000 Oxford units 4/6

This quantity is intended primarily for dilution and local application, but may be used for parenteral injection if desired. (Available also in boxes holding 6 ampoules.)

PENICILLIN-SULPHANILAMIDE POWDER

FOR LOCAL APPLICATION. NOT FOR HYPODERMIC INJECTION.

1 bottle containing 10 grammes, 14/6.

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